CWL-H00660-17-1

THE ADVANCEMENT OF LEARNING

BRAIN:

A JOURNAL OF NEUROLOGY.

VOL. XVII.

BRAIN

A JOURNAL OF NEUROLOGY

EDITED
FOR THE NEUROLOGICAL SOCIETY OF LONDON
BY

A. DE WATTEVILLE

VOL. XVII.

UNIVERSITY OF
LIBRARY
ADVANCEMENT OF LEARN

London:
MACMILLAN AND CO.
AND NEW YORK.
1894.

P16242

LONDON
PRINTED BY JOHN BALE & SONS,
87-89, Great Titchfield Street, W.

UNIVERSITY OF CALCUTTA
LIBRARY
THE ADVANCEMENT OF LEARNING

CONTENTS.

ORIGINAL ARTICLES AND CLINICAL CASES :—

PAGE
Recent Work on the Cerebellum and its Relations, with Remarks on the Central Connexions, and Trophic Influence of the Fifth Nerve. By D. Ferrier, M.D., F.R.S. 1
On Hæmatoma of the Dura Mater associated with Scurvy in Children. By G. A. Sutherland, M.D., M.R.C.P. 27
Observations on a Chinese Brain. By C. Hubert Bond, M.B. ... 37
On General Paralysis (with a Critical Digest). By W. Julius Mickle, M.D., F.R.C.P.Lond. 50
A Case of Pseudo-bulbar Paralysis, due to Lesions in each Internal Capsule; Degeneration of Direct and Crossed Pyramidal Tracts. By W. S. Colman, M.D., M.R.C.P. 86
Imperative Ideas. By D. Hack Tuke, M.D., F.R.C.P., LL D ... 179
On the Association of Disease of the Spinal Cord with Pernicious Anæmia. By H. M. Bowman, M.D.... 198
Pathology and Treatment of Graves' Disease. By James J. Putnam, M D. (Harvard) 214
Peripheral Neuritis in Exophthalmic Goitre. By Arthur Maude ... 229
A Classification of Reflex Actions. By D. Fraser Harris, B.Sc. (Lond.), M.B., C.M. (Glasgow) 232
Case of Tumour of Pons and Left Crus Cerebri. By Seymour J. Sharkey, M.D., F R.C.P. 238
On Disturbances of Sensation, with Especial Reference to the Pain of Visceral Disease. (Part II. : Head and Neck). By Henry Head, M.A., M.D.... 339
Local Anæsthesia as a Guide in the Diagnosis of Lesions of the Upper Portion of the Spinal Cord. By M. Allen Starr, M.D., Ph.D. 481
The Finer Anatomy of the Infundibular Region of the Cerebrum, including the Pituitary Gland. By Henry J. Berkley, M.D. Baltimore 515

PAGE
On Some of the Newer Aspects of the Pathology of Insanity. By W. Lloyd Andriezen, M.D.Lond. 548

PAGE
Introductory 548
Bases for Study 550
Old Views 552
Comparative Neurology ... 553
The Cerebral System ... 567
The Cortex 569
The Work of Golgi, &c. ... 572
Material for Present Work 573
Cortical Lamination ... 574
First Layer 578
Pyramidal System... ... 585
Olfactory Projections ... 590
Ambiguous Cell-System ... 594
Long Pyramidal System ... 598
Regional Variation ... 604
Cortical Sensory Mechanisms 605
Conclusions of Part I. ... 606
Part II. 607
Organisation of Visual Area 608
Primary Sensorial Neurons of Cortex 613
Polymorphic System ... 614
Amphibian Brain 614
Reptilian Brain 616
Foreshadowing of the Mammalian Brain ... 620
Meynert's Association System 621
Greater Extent of Connections 622
Evolution of Polymorphic Cell Types 623
Migration of Sub-Cortical Cells 625
Comparative Growth of the Polymorphic System ... 626
Fields of Conjunction ... 627
Loss of Insulation in such Fields 628
New Activities in such Fields 628
Sensory Sequelæ 632
Specific Sensations ... 632
Jacksonian Epilepsies ... 632
Psychical Epilepsy ... 634
Sensory Processes in the Rolandic Area 635
Results of Visual Excitations 636
Aphasia 638
Anatomico - Physiological Evolution of Rolandic Cortex 638
Quantitative Evolution ... 639
Qualitative Evolution ... 640
Physiological Elaboration... 642
Education 643
Quality and not Quantity... 644
Law of Psychogenesis ... 645
Language — Co-operation in Brain Development ... 646
Further Mental Evolution 648
Pathogenesis 649
(*a*) Protective Mechanisms in the Brain ... 650
Ependyma Formations ... 656
(*b*) Nutritive Mechanisms 660
Bases of Pathogenesis, *i.e.*, the Insanities 664
Alcoholic Insanity of Slow Growth 665
Generalised and Extensive Type of Onset 667
Clinical Symptomatology... 667
Internal Pathology ... 668
Correlation of Clinical with Pathological 672
Clinico-Psychological ... 675
"Motor" Symptoms ... 678
Loss of Power 678
Insomnia 680
Pathology 680
Melancholia 682
Pathology 683
Special Heredity 685
General Conclusions ... 687

PAGE
Observations upon the Central Relations of the Vago-Glosso-Pharyngeal, Vago-Accessory and Hypoglossal Nerves, from the Study of a Case of Bulbar Paralysis. By William Aldren Turner, M.D.Edin., M.R.C.P.Lond., and William Bulloch, M.D.Aberd. 693

CRITICAL DIGESTS :—

Hysteria and Neurasthenia. By J. Michell Clarke, M.A., M.D., M.R.C.P.

I. Papers on the Theory of Hysteria and of Neurasthenia ... 120
II. Papers on Miscellaneous Facts Concerning, and Symptoms of, Hysteria 145
III. Papers on Hysterical and some other Functional Disorders of Motion 165
IV. Papers on Hysterical Disorders of the Organs of Vision ... 263
V. Papers on Hypnotism, Hysterical Somnambulism and Double Consciousness 272
VI. Papers on the Association of Hysteria with Organic Nervous Disease 280
VII. Papers on the Traumatic Neurosis 286
VIII. General Features of Neurasthenia (Löwenfeld)... ... 297
IX. Treatment of Hysteria and Neurasthenia (Lowenfeld) ... 310
On Exophthalmic Goitre. By A. Maude 246

REVIEWS AND NOTICES OF BOOKS :—

Wilhelm Wundt. Grundzüge der Physiologischen Psychologie.—Oswald Külpe. Grundriss der Psychologie, auf Experimentelle Grundlage dargestellt. (E. B. Titchener) 90
Th. Flournoy, M.D. Métaphysique et Psychologie. (Shadworth H. Hodgson) 103
Th. Flournoy, M.D. Des Phénomènes de Synopsie (Audition Colorée) : Photismes ; Schèmes visuels ; Personnifications (James Sully) 115
Tucsek's "Pellagra." (Taylor) 513
Obersteiner's "Arbeiten." (Bulloch) 514

ABSTRACTS OF BRITISH AND FOREIGN JOURNALS :—

Prof. Gaule on the Trophic Properties of Nerves 322
Wertheim Salomonson on Tremor. (Dr. Voorthuis) 337
Henschen on the Anatomy of the Visual Path 710

LIST OF CONTRIBUTORS.

ANDRIEZEN, W. LLOYD, M.D. (Lond.)
BERKLEY, HENRY J., M.D.
BOND, C. HUBERT, M.B.
BOWMAN, H. M., M.D.
BULLOCH, WILLIAM, M.D. (Aberd.)
CLARKE, J. MICHELL, M.A., M.D., M.R.C.P.
COLMAN, W. S., M.D., M.R.C.P.
FERRIER, D., M.D., F.R.S.
HARRIS, D. FRASER, B.Sc. (Lond.), M.B., C.M. (Glas.)
HEAD, HENRY, M.A., M.D.
HODGSON, SHADWORTH H.
MAUDE, ARTHUR
MICKLE, JULIUS, M.D., F.R.C.P. (Lond.)
PUTNAM, JAMES J., M.D. (Harv.)
SHARKEY, SEYMOUR J., M.D., F.R.C.P.
STARR, M. ALLEN, M.D., Ph.D.
SULLY, JAMES, M.A., LL.D.
SUTHERLAND, G. A., M.D., M.R.C.P.
TAYLOR, JAMES, M.D.
TITCHENER, E. B., B.A., Ph.D.
TUKE, D. HACK, M.D., F.R.C.P., LL.D.
TURNER, W. ALDREN, M.D. (Edin.), M.R.C.P. (Lond.)
VOORTHUIS, DR.

LIST OF ILLUSTRATIONS.

PAGE.

Recent Work on the Cerebellum and its Relations; with Remarks on the Central Connexions, and Trophic Influence of the Fifth Nerve. Plate 32

Observations on a Chinese Brain. Figs. 1-3 ... ...42, 43

Hysteria and Neurasthenia. Chart 160

On the Association of Disease of the Spinal Cord with Pernicious Anæmia. Figs. 1-8 202

Case of Tumour of Pons and Left Crus Cerebri. Diagram. Figs. 1, 2... ... ... 240, 243, 244

On Disturbances of Sensation, with Especial Reference to the Pain of Visceral Disease. Figs. 1-61. Plates I. and II., 346, 348, 350, 352, 353, 355, 356, 358, 359, 361, 363, 364, 366, 367, 369, 371, 373, 374, 375, 376, 377, 378, 380, 381, 382, 383, 384, 387, 389, 393, 402, 405, 411, 412, 415, 420, 421, 422, 423, 426, 428, 432, 439, 443, 446, 448, 450, 453, 455, 458, 469, 470, 471, 472, 473, 474

Local Anæsthesia as a Guide in the Diagnosis of Lesions of the Upper Portion of the Spinal Cord. Figs. 1-12. Plates I. and II. 484, 485, 487, 489, 491, 492, 493, 494, 495, 498, 502, 506

The Finer Anatomy of the Infundibular Region of the Cerebrum, including the Pituitary Gland. Figs. 1-30 546

On Some of the Newer Aspects of the Pathology of Insanity. Figs. 1-36, 552, 555, 556, 560, 575, 577, 579, 582, 583, 584, 586, 589, 591, 595, 596, 597, 602, 613, 615, 617, 619, 629, 631, 640, 650, 652, 654, 656, 658, 659, 660, 662, 663, 669, 670, 684

BRAIN.

PART I., 1894.

Original Articles.

RECENT WORK ON THE CEREBELLUM AND ITS RELATIONS; WITH REMARKS ON THE CENTRAL CONNEXIONS, AND TROPHIC INFLUENCE OF THE FIFTH NERVE.[1]

BY DAVID FERRIER, M.D., F.R.S.

GENTLEMEN,—In assuming the office of President for the ensuing session, I desire, in the first place, to express my high appreciation of the distinguished honour, and to say that it will be my endeavour to promote to the utmost the best interests of the Society, and to maintain, with your help and co-operation, the high standard which the discussions and communications to the Society have attained.

It seems appropriate that on this occasion, the opening day of a new session, we should, as a branch of the universal brotherhood of science, pay some tribute to the memory of one recently passed away, whom we all, without exception, acknowledge to have been one of the greatest masters of our science in this or any age—the late Professor Charcot. It is altogether unnecessary that I should, before such an audience, attempt to recount the services which Professor Charcot has rendered to Neurological science. There is scarcely a subject which he has not touched, and which he has not likewise adorned. His death, ripe in years and wisdom, and in the full enjoyment of his splendid reputation, has deprived the world of one whose place can never be filled, and our Society of one of the most illustrious of its small and select list of honorary members. Professor Charcot took much interest in our Society, in which he had so many personal friends, and on the last occasion when I saw him, shortly before his death, he promised to be present and take an active part at one of our meetings during the ensuing session. This, alas! to our grievous disappointment, was not to be. During the last year the Society has also lost in Dr. James Anderson, one of its office-bearers, esteemed alike for his qualities both of head and heart, and just as he was beginning to reap the fruits of the solid reputation he had established for careful and accurate clinical work; and in Dr. Walter Hadden, a neuro-pathologist of brilliant promise, an active participator in the discussions of the Society, and frequent contributor to its journal.

---

[1] Being the Presidential Address to the Neurological Society, January 25, 1894.

In inaugurating the work of a new session, it has been the custom of my predecessors to introduce some question of more or less present interest in Neurology in one or other of its various aspects. In accordance with this custom, which I consider well worthy of imitation, I have chosen as the chief subject of my discourse this evening one which, though old and worn, one would think, threadbare, is yet ever new and apparently as far as ever from being exhausted: to wit, the functional and anatomical relations of the cerebellum.

These have been practically reconstructed by the recent work of Luciani[1] and Marchi, and it is more particularly in reference to the views expounded by these enquirers, that I wish to engage your attention and consideration for a brief space. I have selected this topic from others that offered themselves, for the special reason that it has been my latest study, and has formed during the last two years a joint research with my colleague Dr. Turner. (*Proc. Roy. Soc.*, Dec. 14, 1893.)

---

[1] "Il Cervelleto," Firenze, 1891; *German Trans.*, Kleinhirn, 1893.

Luciani is the first who has, by skilfully contrived and well-executed experiments, succeeded in studying for long periods of time the symptoms consequent on complete or partial removal of the cerebellum in the higher animals; viz., dogs and monkeys; whereas, with the exception of fowls and pigeons, most previous experimenters had limited their observations to the more immediate effects of such lesions. But these, as the researches of recent years have more and more strongly emphasized, are of themselves inadequate data, and may even lead to erroneous conceptions as to the true functional relationships of any particular organ or nerve centre. The importance of Luciani's work cannot be overestimated, and his analysis and synthesis are worthy of the most careful consideration.

The immediate effects of cerebellar lesion, as described by Luciani, and amply confirmed by our own experiments, do not differ materially from those which are familiar to everyone in the classical descriptions of Flourens. The stationary phenomena, that is, the phenomena of deficiency, are those which are particularly illustrated by abundant richness of experimental detail. By overwhelming proofs, if further proof were necessary, he has disposed finally of the supposed relation of the cerebellum to the sexual instinct. Indeed, as he himself says: "In the history of my experimental animals, there were so many love episodes, of such a wanton and grotesque character that one might really ask whether the loss of the cerebellum was not the cause, perhaps indirectly, of an abnormal degree of licentiousness; and these love relationships were not without their natural consequences. On the contrary, pregnancies, lyings-in, and parturitions followed each other so frequently that my laboratory appeared to have been converted into a maternity hospital."

There is no proof that the cerebellum has any share, or takes any part in psychical manifestations. The total removal of the cerebellum causes no discoverable impairment of any of the special senses, or of any of the forms of cutaneous or of muscular sensibility. In this respect our experiments are entirely in harmony with his.

Luciani has also furnished additional and conclusive evidence to show that the influence of the cerebellum is direct, and not crossed, and that each half of the cerebellum exerts its influence mainly, if not exclusively, on the corresponding side of the body.

His experiments, with which our own entirely agree, have summarily disposed of the theory of Nothnagel that to the middle lobe alone belong those functions which are commonly ascribed to the cerebellum, and that on lesion of this lobe, directly or indirectly, the symptoms of cerebellar disease exclusively depend. This view, which has always appeared to me a very strained reading of the facts of disease, in accordance with a preconceived notion founded on imperfect data, has been shown by Luciani's experiments, as well as our own, to be without foundation; for the effects of removal of the middle lobe are not more pronounced, and are not more enduring than those which follow extirpation of the lateral lobe.

When the cerebellum has been totally removed from monkeys—an operation which we have found to be a most formidable one, and more frequently fatal than successful—the animals exhibit the most tumultuous disorders of equilibrium, so that station and locomotion are, for the time, altogether impossible. Gradually, however, these tumultuous disorders subside to such an extent that the animal is able to sit up, with the help of some support to which it clings, and ultimately regains such a degree of stability that it may dispense with extraneous aid, but is so tottery that it falls over on the slightest disturbance or excitement. Its gait is of a peculiar sprawling character; at first, barely raising its abdominal surface from the ground, and planting its limbs wide apart so as to increase the basis of support; these being in progression raised and set down in a brusque and characteristically ungraduated fashion, which character the movements of the limbs retain for an indefinite period. Notwithstanding this extraordinary unsteadiness and instability of equilibrium, the animals are, however, able to grasp tenaciously with both hands and feet, so much so that it is sometimes more easy to drag the chair to which they may cling than loosen their grip; and they are able to climb a rope with agility hand-over-hand, in no way differing in this respect from perfectly normal animals. But the most noteworthy and persistent feature is the remarkable astasia or unsteadiness of the head, trunk, and limbs, which are either agitated by constant fine tremors, apart from obvious muscular exertion, or exhibit oscillations of the typical disseminated sclerosis type on volitional exertion; so that, for instance, an attempt to lay hold of a piece of fruit excites such wild oscillations of the hand and arm that the object aimed at is either knocked away, or, if seized, after many unsuccessful efforts, is with the utmost difficulty brought up and held to the mouth. This instability is a persistent feature, and, though lessening to some extent, never entirely disappears, and was clearly evident in one of Luciani's monkeys a whole year after the operation.

If only the lateral lobe, or one half of the cerebellum, is removed, the persistent symptoms, after the cessation of the primary disorders, namely, the sprawling, ungraduated action of the limbs, and the tremors or instability on volitional effort, are confined to the same side as the lesion.

If, on the other hand, the middle lobe is destroyed, or extensively injured, as by antero-posterior division, the symptoms are essentially of the same character as those which follow destruction of the whole organ, but they do not affect one side more than the other, and, according to our observations, are more pronounced in the head and trunk than in the limbs. They also appear to be less persistent, and, in the course of a few months, pass off to such an extent that, except on careful examination, it would be difficult to distinguish an animal so operated upon among its normal companions.

In the course of our investigations, Dr. Turner and I have succeeded in dividing the individual peduncles of the cerebellum with comparatively little injury to neighbouring structures at all calculated to complicate the symptomatology. When the primary disturbances, which I will allude to subsequently, have passed off, the persistent phenomena—whichever peduncle is cut—are essentially of the same character as those following destruction of the lateral lobe, and are confined to the limbs on the side of lesion.

Such is a brief outline of the symptoms, temporary and permanent, following total or partial extirpation of the cerebellum and section of its peduncles. The question is, how are these to be explained in accordance with the anatomical connexions and relationships of the cerebellum? In answer to which I fear that, notwithstanding the valuable researches of Luciani, the opinion of Vulpian that "the problem of the functions of the cerebellum is still far from being definitely solved" is not inapplicable to the present state of our knowledge. Luciani's views are, briefly, as follows:—

The cerebellum, with its annexes, constitutes a more or less independent system. It is not an organ interpolated in the course of the cerebro-spinal tracts, but is an "end-organ," or, so to speak, an appendage to the cerebro-spinal axis, directly or indirectly related to certain peripherical sensory organs, and in direct efferent relationship with certain ganglia of the cerebro-spinal axis, and indirectly with the motor apparatus in general. It is functionally homogeneous, each part exercising the functions of the whole, but having special relation to the muscles on the corresponding side of the body. The symptoms following its destruction are, in the first place, *irritative* phenomena, opisthotonic or pleurosthotonic in character, according as the lesion is general or unilateral, together with rotation round the longitudinal axis, and squinting, or deviation, of the optic axis; secondly, *ataxic* phenomena, or phenomena of deficiency (Ausfallserscheinungen), which are more or less compensated by volitional effort on the part of the animal (functional compensation), or by such parts of the organ as have escaped destruction (organic compensation). The phenomena of deficiency, or cerebellar ataxy, are ascribed by him to three principal conditions, which he has a special fondness for reiterating; to wit, *asthenia*, *atonia*, and *astasia*, due to the loss of a sthenic tonic, and static influence, which, according to him, the cerebellum normally exerts on the apparatus of movement, and which is the essence of its function. In addition to this, he ascribes to the cerebellum a trophic function, direct on the efferent tracts which spring from it, and which, therefore, degenerate when the cerebellum is destroyed, and indirect on the nutrition of the body generally.

I should like, with your permission, to examine some of these views a little more in detail.

Touching the so-called irritative nature of the primary disturbances (Reizerscheinungen), which Luciani ascribes to inflammatory action set up by the lesion ("Kleinhirn," p. 151), I am persuaded that this is erroneous, and would prefer with Goltz to term them dynamic, or Hemmungserscheinungen, without attempting to offer a more exact explanation of the mode in which they are brought about. They certainly occur under conditions which entirely exclude inflammatory action, and under methods which, when applied to the cerebral cortex, cause paralytic and not irritative symptoms; and, moreover, there is reason to believe that the phenomena are entirely reversed when the conditions are such as to excite true vital irritation or inflammatory action. The so-called irritative phenomena occur, according to Luciani, more especially when the peduncles are injured, and this is, on the whole true; for, according to our experiments, rotation round the longitudinal axis certainly occurs more constantly when the peduncles are divided than when the lateral lobes are removed. But this is not an absolute rule, and one of the most marked instances of rotation round the vertebral axis which we observed was an animal in which only the cortex of the lateral lobe had been partially cauterized, and under conditions likely to excite irritation of neighbouring parts. In this case the rotation was exactly in the opposite direction to that which usually occurred. The direction of the rotation appears to be the subject of some confusion and difference of opinion. Magendie, as is well-known—and his observations have been confirmed by Hitzig and others—described the rotation after section of the middle peduncle as taking place towards the side of lesion; so that, for instance, if the left were cut the animal would roll to this side with incredible force and rapidity. Luciani, however, describes the rotation as uniformly occurring from the injured to the sound side, so that, *e.g.*, if the right side of the cerebellum is removed, the rotation takes place from right to left.

Our experiments on the cerebellar peduncles, effected in a precise manner by simple section, are absolutely in harmony with the results obtained by Magendie and Hitzig. We have found that, without exception, the animals roll towards the side of lesion.

The apparent discrepancy between our results and those of Luciani has been so uniform that we cannot but think that it is more in the manner of description than in the actual facts themselves. The facts, according to our observations, are that the animals, when placed on the ground, roll towards the side of lesion, as if carried by some irresistible force. And yet the impulse which causes the animal to roll towards the side of lesion, may be correctly described as a rotation round the vertebral axis from the injured to the sound side. Thus, if the left peduncle is divided, the animal will roll towards the left, but the impulse which causes this rolling movement would, if the animal were viewed in front, appear as a rotation round the vertebral axis from left to right. It might, therefore, be equally correct to say that the *rotation* is from the injured to the sound side, and that the *rolling* is towards the side of lesion. That this is the real explanation of the apparent discrepancy, I gather from a passage in which Luciani gives a detailed account of what he terms rotation from left to right. He says ("Kleinhirn," p. 139): "When the animal [a dog] was laid on its left side the torsion of the vertebral column was such as to bring the dorsal surface downwards, and the feet in the air. From this position, and with movements of all four limbs, it turned on its right side with its back to the right, and then on to its left side." It appears to me evident that if these movements were repeated several times in succession the result would be that of an animal rolling towards the left side. If this is not the true solution of the seeming discrepancy as to the direction of the rotation, as described by

Magendie and Luciani, we should say that our results are in agreement with those of Magendie, and that the direction in which animals roll after removal of the cerebellar hemisphere, or section of its peduncles, is towards the side of lesion.

The deviations of the optic axes, described by Magendie in connection with the rotatory phenomena, we have found in monkeys to be far from uniform, and we have not been able to establish any constant relation between lesion of any particular portion of the cerebellum, and any particular distortion or deviation of the optic axes. Nor have we found those indications of tonic contracture of the limbs in monkeys on the side of lesion such as Luciani appears to have uniformly observed and recorded as signs of irritation.

A special and characteristic attitude is assumed by animals after section of one or other peduncle, namely, curvation of the vertebral column, with the concavity towards the side of lesion, adduction and flexion of the limbs on the same side, and abduction and extension of the limbs on the opposite. The chin, as a rule, deviates towards the sound side, that is, in the direction of the rotation which causes the animal to roll towards the side of lesion. But the flexion of the limbs on the side of lesion is not a rigid flexion or contracture, and offers no resistance to passive movements or change of position.

Let us now examine what Luciani considers to be the essential factors of cerebellar ataxy, namely, asthenia, atonia and astasia.

First, as to asthenia, by which is meant a defect of energy, due to the loss of some influence supposed to be exerted by the cerebellum, which heightens or reinforces the energy of muscular contraction. Of the existence of any such influence, such as has been contended for by Luys, Weir-Mitchell and others, or that the phenomena of cerebellar ataxy can in any way be ascribed to such a condition, I have discovered no satisfactory evidence, either from a study of Luciani's experiments, or in the facts of our own, or in the phenomena of cerebellar disease in man. On the contrary; clinical observation has abundantly shown that in cases of atrophy or disease of the cerebellum, of such a nature, and of such extent; as to render station and locomotion altogether impossible, the patients may in the recumbent position perform all movements of the limbs with energy and vigour. Of one of his patients, who was suffering from atrophy of the cerebellum, and who could not walk except in the most bizarre fashion, pushing a chair in front of her and often falling down, Vulpian remarks: "Yet, and this is very remarkable, she retained such muscular vigour that when one held out a hand to save her from falling she gripped so firmly as to cause pain."

When a monkey, within a few days after complete removal of its cerebellum, and so unstable as to be unable to sit up without support, or progress except in a sprawling fashion with its abdomen in contact with the ground, is, notwithstanding, able to climb a rope or lattice work hand-over-hand with apparently the same agility as a normal monkey, and grips so firmly with hands and feet as to require considerable force to detach it, I do not think the term "asthenia," or defect of muscular energy, is at all applicable to such an animal. And when a monkey, within an hour of removal of the left lobe of the cerebellum, and still unable to sit up, is able to grip so firmly with its left hand as to bear the whole weight of its body on it, I do not think that its left limbs can at all properly be described as being affected by asthenia. Yet these and similar facts might be multiplied from our observations.

In no case in which either the lateral lobe was removed, or a peduncle cut, could we detect any appreciable difference in the grip of the two sides. We found it, however, difficult to make exact dynamometric observations, as the animals were not always in the humour to make that trial of their strength which alone would permit of accurate comparison, and without which tests of this kind are altogether fallacious. There is, however, no doubt that an animal after removal of one lateral lobe prefers to use the limbs on the sound side, but this is obviously more in consequence of the instability of the limbs than from any real want of strength, and such, in my opinion, is the real explanation of the apparent want of strength in the limbs on the side of lesion which Luciani has so frequently noted. For the most part, he speaks of a semblance of weakness only, as evidenced by the doubling up of the limbs and tendency to fall on the injured side. But when the same monkey which exhibits this defect is able to climb well, and a dog to swim vigorously, as he admits, the symptom cannot be explained by loss of muscular energy.

One of the proofs relied on by him as indicating relative weakness of the limbs on the side of lesion is of so remarkable a character as to deserve special mention. Thus, he says that dogs deprived of one-half of the cerebellum, when swimming, tend, almost invariably, to the opposite side. Thus, if the right half of the cerebellum has been removed, the animal swims towards the left, in consequence, he says, "of the greater energy of movement of the limbs on the left side." ("Kleinhirn," p. 171.) Any swimmer, however, would say that in order to swim to the left he would require to make more vigorous efforts with his right limbs, and the very fact mentioned by Luciani would appear to prove exactly the opposite of what he says. In further corroboration of this may be mentioned another observation of his, viz., a dog in which the right side of the cerebellum had been removed, together with the motor centres (sigmoid gyrus) of the left hemisphere, thereby causing paresis of the right limbs. This dog in swimming, swam towards the right side, because, as he says, and truly, "the left limbs struck the water more vigorously than the right." (*Ibid.*, p. 133.)

With these remarks I would dismiss Luciani's theory of asthenia as an element in the causation of so-called cerebellar ataxy, as being unsupported by valid evidence. That animals after complete removal of the cerebellum may, as Weir-Mitchell says, show signs of exhaustion after sustained muscular effort sooner than normal animals, is, I believe, more easily explained on my own hypothesis that the efforts at functional compensation, effected, as Luciani has verified, by the cerebral motor centres, are a considerable strain on the animal's power, and, in consequence, lead to exhaustion much sooner than under normal conditions.

Next, as to the evidence of atonia, or loss of tone in the muscles. The usual test of the tone of muscles is the readiness, or otherwise, with which they respond to mechanical tension, such as is constantly exemplified in the usual method of eliciting the patellar-tendon reaction. Luciani has, however, apparently not investigated the condition of the tendon reflexes in his experimental animals. As a proof of the loss of tone, he speaks of the muscles of the limbs on the side of lesion as appearing to him more flaccid, and of the leg occasionally giving way, as if the tone suddenly ceased, &c., all of which appear to me more matters of constructive speculation than actual realities.

That the tone of the muscles, so far at least as indicated by the tendon reactions, is not diminished by cerebellar extirpation, complete or unilateral, or after section of its peduncles, has been amply proved by our own experiments, as well as by those of Dr. Risien Russell. (*Proc. Roy. Society*, vol. liii., p. 430.) We have not, however, observed the marked exaggeration of the knee-jerk on the side of lesion, which Dr. Russell appears to have found in dogs. As a rule, little or no difference could be made out after extirpation of the lateral lobe, or section of the peduncles on one side, immediately after the operation. If anything, the difference was in favour of the reaction on the side of lesion, but it showed a distinct tendency to increase the longer the animals survived; and when the whole cerebellum was extirpated, the knee-jerks were, after some months, decidedly increased, as compared with the normal, but there was no rigidity of the limbs.

These facts lead me to diverge for a moment to comment on the views propounded by Drs. Hughlings Jackson and Bastian as to the mechanism of the development of rigidity in limbs paralysed by transverse lesions of the spinal cord. Dr. Bastian has adduced weighty evidence (*Med. Chir. Trans.*, vol. lxxiii., p. 151), and he has been amply supported by Bowlby (*Ibid.*, p. 313), and others, to show that in total transverse lesions of the spinal cord the tendon reflexes are abolished and never return, and that this condition cannot be satisfactorily explained on the hypothesis of a coincident lesion, or mere dynamic disturbance of the centres situated below the lesion. Though I think the point is one which may well bear further investigation, I am prepared, for the purposes of argument, to admit Dr. Bastian's law as being universally applicable. But his further proposition to the effect that the development of rigidity in limbs paralysed from cerebral disease, or partial lesion of the cord, is due to the "unrestrained," or, as Dr. Jackson would say, the "unantagonized" influence of the cerebellum passing downwards, perhaps through the grey matter of the cord, I cannot allow to pass without question.

First, I can find no experimental proof that the cerebellum exercises any such influence as that contended for by Dr. Bastian. It is no doubt true that in some cases of disease in the posterior fossa, involving the cerebellum, the tendon reflexes are abolished. I am not acquainted with, and am myself unable to offer any satisfactory explanation of this fact, but, certainly, it is not the rule that the knee-jerks are abolished in cerebellar disease. And when we find that in monkeys, after complete removal of the cerebellum, the knee-jerks instead of being lost become actually increased, it is clear that the mechanism for the increase of the knee-jerks still exists. That this is independent, both of the cerebrum and cerebellum, is further evident from the fact that in monkeys the knee-jerks may become increased, and late rigidity ensue, when the spinal cord has been totally divided transversely. In illustration of this point, I will here briefly mention the facts of two experiments made by Dr. Turner and myself.

In the first case, the spinal cord was divided transversely and completely at the level of the eighth dorsal root in March, 1891. Immediately after the operation the knee-jerks were capable of being elicited as before the operation. The animal exhibited the usual paraplegic symptoms, both as regards total paralysis of motion and sensation, and continued so until its death on July 24, that is, fully four months. During this period there gradually developed well-marked increase of the knee-jerks, as well as of the superficial reflexes, together with considerable rigidity of the limbs. The animal was exhibited before its death to Drs. Hughlings Jackson and Bastian, who both satisfied themselves as to the condition here described.

In a second case, the cord was completely divided at the level of the sixth dorsal nerve on July 21, 1891. Both knee-jerks were readily elicited immediately after the operation, if anything, more markedly than before. The animal lived till September 29 ; that is, two months. This animal, however, exhibited some noteworthy differences as compared with the former. Instead of a gradual increase of the knee-jerks and development of late rigidity, no reaction could be obtained on August 4, that is, twelve days after the operation. The knee-jerks proper remained in abeyance till the 20th, that is, for sixteen days. During this time the muscles of the limbs had undergone considerable wasting, and the faradic contractility of the quadriceps extensor became decidedly diminished as compared with the other muscles, and more so on the left than on the right. After the 20th the right knee-jerk re-appeared and continued to increase. The left remained in abeyance until September 10 at least, after which date the knee-jerks were not examined, as the animal died during the holiday season.

The first of these two cases shows conclusively that in the cord itself there exist the conditions for the presence and increase of the knee-jerks, and the subsequent development of rigidity or contracture of the limbs. And the second case shows also, in a highly instructive manner, that in the cord itself conditions may occur which, either temporarily, or it may be permanently, prevent the manifestation of these phenomena. In both these cases, the influence of the cerebrum and cerebellum was, from the first, entirely excluded. But it will probably be said that though this may be true of monkeys, it is not applicable to man. That the symptomatology of total transverse lesions in man and the lower animals is widely different, I readily admit. Indeed, we have every reason for believing that the independence of the spinal centres is much greater in the lower animals than in man. We all know the extraordinary adaptiveness and apparent intelligence of the spinal cord of a frog; and the experiments of Goltz on dogs have shown that, even in animals so high in the scale as dogs, the hind limbs, after total transverse section of the spinal cord, remain capable of a number of the most complex, reflex or responsive actions. In answer to some enquiries I made of him in regard to his animals, Professor Goltz wrote to me: "Several of my dogs learnt to stand on their hind legs, and even to walk a few steps. While so standing, the hind legs became stiff in consequence of a general contracture of the muscles of a reflex character, conditioned by the position of the animal." Such things are, of course, impossible in monkeys and, *a fortiori*, in man.

The point, however, which I desire to emphasize is, that the increase of the knee-jerks, and development of rigidity, can occur in monkeys altogether independently of the cerebellum, owing to conditions in the cord itself, in which also the opposite conditions may occur, either temporarily or permanently. The difference between the symptomatology of total transverse lesions of the cord in man and the lower animals is, in my opinion, due to the greater independent vitality of the spinal centres in the latter as compared with the former. In these, total separation from the higher centres leads to such impaired vitality, that they are no longer capable of manifesting that increased excitability, which is the primary cause of the phenomena in question.

Let us now return to the third cardinal factor of cerebellar deficiency; namely, the condition which Luciani terms "astasia." This term very aptly describes the primary instability, both of station and locomotion, as well as the subsequent, and more persistent, tremor and unsteadiness, not only of the body as a whole, but of the limbs, which is so noteworthy a feature after cerebellar extirpation in monkeys and dogs. The reeling gait and the various degrees of what most nearly represents alcoholic intoxication, are familiar and generally recognised symptoms of cerebellar disease in man, but the tremor and unsteadiness of the limbs, though occasionally noted, and more particularly in cases of cerebellar atrophy, are not so common, and yet they are sometimes present, as I have myself had occasion to observe. When they occur more particularly on one side, they ought to furnish a reliable guide to the determination of the position of the lesion, inasmuch as we have seen that they occur specially on the side of lesion. It would appear, therefore, that the instability of cerebellar defect is not limited to the muscles of the trunk, but affects the muscles, in general, and manifests itself not only in the unsteadiness on volitional effort, but also in the ungraduated or dismetric (Luciani) manner in which the limbs are raised and planted in progression, in spite of the most complete compensation which the voluntary motor centres are capable of effecting. But the term "astasia" is, after all, only a description, better perhaps than any other that has been applied to the phenomena of cerebellar deficiency, and not an explanation; and the problem still remains, how is the astasia conditioned? To explain this is to my mind to explain the true mechanism of the cerebellum.

Luciani sees in the tremors and oscillations, &c., a defective summation of the individual impulses on which the muscular contraction depends, and he looks on the abruptness of the movements of the limbs as due to a sudden flaccidity (Erschlaffung), of the antagonists while the flexors or extensors are acting respectively; or, it may be, owing to collateral muscles intervening which are not generally concerned in the action in question. And there is much to be said in favour of a hypothesis of this kind. And yet I question whether this is any improvement on the hypothesis of Herbert Spencer, that the cerebellum is the organ of "doubly compound co-ordination in space," while the cerebrum is the organ of "doubly compound co-ordination in time," so often and so ably expounded and applied by Dr. Hughlings Jackson. "According to this hypothesis," as James Ross has lucidly stated it, "all the muscles of the body are innervated both by the cerebrum and cerebellum, but in an inverse order. The cerebellum regulates the muscular contractions necessary for the maintenance of our attitudes in space, while the cerebrum regulates the contractions necessary to effect all the change of attitude which are made in response to the successive impressions occurring in time. Now, so long as a particular attitude is maintained in opposition to gravity and other forces, the contractions of the various groups of muscles concerned must be continuous and in equilibrium with each other, while each change of attitude necessitates the overthrow of this equilibrium, involving the preponderance of the contractions of some groups of muscles over those of others, so that the change of attitude involves alternate muscular contractions and relaxations. Speaking broadly then, the cerebellum regulates continuous or tonic muscular contractions. It will be seen, therefore, that every compound muscular adjustment necessitates the co-operation of both these organs. No change of attitude can be effected by the cerebrum, except in so far as a certain attitude was previously maintained by the cerebellum, and no steady movement can be produced by the alternate contractions of some groups of muscles except in so far as other groups of muscles are maintained in a state of continuous contraction; hence it may be inferred that all the movements of the body are co-ordinated both in the cerebellum and cerebrum." (Ross, "Diseases of the Nervous System," vol. i., p. 57.) The cessation of the tonic or continuous influence of the cerebellum should, therefore, exhibit itself in an unsteady, ungraduated character of all the motor adjustments, whether of the body as a whole, or of its individual parts, and would, theoretically, produce a state of affairs which agrees well with the actual effects of cerebellar extirpation in animals.

Even, however, if we assume that this is the true formula for the influence of the cerebellum, it still remains to be determined how its activity is called into play and brought to bear on the muscles, either in association with the cerebrum, or independently. That it is a more or less independent responsive mechanism is clearly shown by the fact that the stability of motor adjustments is still capable of being maintained after the cerebrum has been removed. That the cerebellum stands in complex afferent and efferent relations with the cerebro-spinal system is undoubted, but we, as yet, know little as to the true functions of the respective centripetal tracts, and less, perhaps, with regard to the centres and tracts through which it acts centrifugally.

The superior peduncles proper appear to be efferent in function; that is to say, after cerebellar extirpation they degenerate *from* the cerebellum up to the opposite red nucleus and thence to the optic thalamus. On the dorsal aspect of the brachium conjunctivum, however, there is a tract which degenerates *towards* the cerebellum, a tract which the observations of Loewenthal,[1] Mott,[2] Tooth,[3] and our own, prove to be the termination of Gowers' antero-lateral spinal tract. As yet, however, there has been no exact determination of the functions of the red nucleus, or of Gowers' antero-lateral tract.

The middle cerebellar peduncle appears also to be efferent from the lateral lobe of the cerebellum to the opposite nucleus pontis. It is, no doubt, through the intermediation of the cells of the nucleus pontis that the cerebellar hemisphere of the one side is brought in relation with the cerebral hemisphere of the opposite. But here again we are in ignorance as to the true signification of those tracts, which are described as connecting the frontal and temporo-occipital regions of the cortex with the cell groups in question.

The inferior cerebellar peduncle contains both efferent and afferent fibres, *efferent* to the opposite inferior olive, by way of the external arcuate fibres; and *afferent*, viz., the direct cerebellar tract from Clarke's vesicular column, largely also of fibres from the posterior median and postero-external columns of the spinal cord by way of the clavate and cuneate nuclei, and probably also of the vestibular branch of the eighth, and, perhaps, also some of the other cranial nerves. But as to the exact functions of these different afferent tracts to the cerebellum, we know very little. No discoverable loss of sensation ensues when they are divided. In particular, destruction of the clavate and cuneate nuclei, though leading to temporary disturbances of equilibrium, causes no discoverable impairment of any of the forms of cutaneous or general sensibility. And as the great bulk of the fibres which spring from the clavate and cuneate nuclei decussate in the interolivary layer, and ascend as the mesial fillet, which degenerates completely when they are destroyed, it is clear that this is not the path of transmission of sensations proper to the cerebrum. A tract which forms the internal division of the restiform body, and termed by Edinger "the direct sensory cerebellar tract," has been supposed to be the path by which the fifth, eighth, and probably also the vagus and glosso-pharyngeal nerves form connections with the middle lobe of the cerebellum. We have found, however, after extirpation of the middle lobe, that the tract in question degenerates downwards, or in an efferent direction, towards Deiters' nucleus. This is the only tract which appears to degenerate centrifugally from the middle lobe, a fact which shows that this lobe has no direct connection with the efferent tracts, which emerge in the cerebellar peduncles. Beyond those already mentioned, there is no proof of the existence of any other efferent tract from the cerebellum to the spinal cord. Marchi,[1] however, has described a tract which degenerates downwards in the antero-lateral region of the cord after extirpation of the cerebellum, which he derives specially from the middle lobe by way of the posterior longitudinal bundles and fillet. The tract described by Marchi occupies the periphery of the antero-lateral region, with an offshoot posteriorly projecting into the lateral column. Our observations, however, lead us to believe that Marchi's views as to the origin of this tract are erroneous; for in a case of total extirpation of the cerebellum, which lived several months, we found no evidence of any degeneration in the position assigned by Marchi to his tract. And yet in one case of removal of the lateral lobe we found a degeneration in the periphery of the anterior column corresponding in position with the anterior division of Marchi's tract; and in another we found a well-marked descending degeneration in the lateral column in the region of the posterior division of the tract described by him. A further careful examination, however, of these cases, indicated that the degeneration in the anterior column was due to lesion of Deiters' nucleus, which would thus be an internode between the middle lobe and the anterior marginal column of the spinal cord, and that the degeneration in the lateral tract stood in relation to lesion of the lateral fillet, which forms a tract connecting the posterior corpus quadrigeminum with the lateral column of the cord. But while so much obscurity still prevails as to the functional significance of the various afferent and efferent tracts which connect the cerebellum with the rest of the cerebro-spinal system, we are not yet in a position to go much beyond mere speculation as to the mechanism of cerebellar co-ordination. In this some of us have indulged with greater or less plausibility, but I do not propose on this occasion to enter on the field of speculation, as I think we may more profitably wait for the dry clear light of further exact research.

---

[1] *Rev. Méd. d. l. Suisse Romande*, 1885.

[2] BRAIN, vol. xv., 1892.

[3] *Ibid.*

---

[1] *Sul. Orig. e. Decor. dei Ped. Cerebell.*, 1891.

If I may trespass on your patience a little longer, I should like to say a few words on a question which, though not directly related to the main subject of my discourse, has yet formed the subject of some inquiry in the course of our other experiments on the connexions of the cerebellum. Thus, on section of the inferior peduncle, it not unfrequently happened that the immediately subjacent, so-called, "ascending root" of the fifth nerve was wholly or partially severed. The result of lesion of this root was more or less complete anæsthesia of the cornea, and of the region of distribution of the sensory branches of the fifth nerve of the head and face. And by other experiments, not necessary to be detailed here, it was proved that this so-called ascending root is the true sensory root of the fifth nerve, and is, in reality, a descending root springing from the Gasserian ganglion, and passing through the substantia gelatinosa Rolandi towards the cells of the posterior cornu as far, at least, as the origin of the second cervical nerve.

On dividing the superior peduncle, one usually severs also the fibres of the so-called "descending root," which courses underneath this peduncle towards the crescentic layer of cells which surrounds the grey matter of the aqueduct of Sylvius. After such section, as well as after section of the motor root within the pons, but not after section of the sensory root, there followed atrophy of the fibres of this root, and of the crescentic layer of cells above-mentioned. The atrophy, however, was not a degenerative, but a simple atrophy, similar to that which occurs, as has been shown by Bergmann,[1] Darkschewitsch,[2] &c., in the central end of a motor root after division of the trunk. This is the root which has been supposed by some to join the sensory, and by others to join the motor division of the fifth: while Merkel has described it as being the trophic root of the trigeminus. This also is the root which Mendel[3] has described as being atrophied in a case of old-standing facial hemiatrophy. A recent experiment of ours points to the descending root as being the motor root *par excellence* of the fifth nerve. No loss of sensation was caused in the cornea or elsewhere by its division, nor did any trophic disturbance occur in the eyeball during the many weeks which the animal survived. On the other hand, atrophic paralysis ensued in the muscles of mastication.

A point which we kept specially before us in our experiments on the fifth nerve, was the so-called trophic influence of this nerve on the eyeball, a question which is still the subject of dispute, and which has come more recently again into prominence in connection with surgical operations on the Gasserian ganglion, with a view to the relief of inveterate trigeminal neuralgia.

The question is, as to the exact causation of the ulceration of the cornea and subsequent destructive inflammation of the eyeball, which have been so frequently observed both in man and animals after lesion or section of the fifth nerve. Mere anæsthesia, with the consequent loss of the protective

[1] *Obersteiner's Arbeiten*, 1892.

[2] *Neur. Centralblatt*, 1893.

[3] *Neurol. Centralbl.*, 1888.

916242

influence conferred by the adaptive reactions of the eyelids, does not seem capable of explaining the facts, for the eye may be insensible from cerebral lesion, or even from lesion of the fifth nerve itself, and yet show no sign of corneal ulceration or destructive inflammation, though the anæsthetic eye has not been more protected from external influences than the other. An interesting case of this kind has been recorded by Hutchinson (*Ophthal. Hosp. Reports*, vol. iv., p. 191), and many others might be alluded to. And the acute inflammation has been seen in cases of lesion of the fifth nerve, not sufficient to cause complete anæsthesia; facts which have led Meissner and others to postulate the existence of a separate set of fibres exercising a trophic influence on the eyeball, apart from those which confer sensibility on it. Nor can the inflammation be satisfactorily accounted for by mere vascular paralysis, for the inflammatory disorders first show themselves in the cornea, the extra-vascular part of the eyeball; and Sinitzin states, though in this he is opposed by Eckhard,[1] that section of the cervical sympathetic, and consequent vaso-motor paralysis, actually prevents the inflammatory consequences otherwise following section of the fifth. Snellen found that when the eyeball was completely protected no inflammation occurred; and he, therefore, attributed the whole of the phenomena to external influences. But these conclusions have been contradicted by others (Decker, Laborde, Büttner, &c.) who have observed speedy destructive inflammation follow section of the nerve in spite of protective precautions; and also cases in which the eye did not become inflamed, though no special precautions were taken to protect it. There remains, therefore, some factor requiring elucidation which is of considerable importance from a practical point of view.

Magendie first pointed out, and in this he was confirmed by Longet and others, that the effects of section of the fifth nerve differ according as the lesion was made behind or in front of the Gasserian ganglion. Thus, whereas, section between the ganglion and the pons did not, or rarely, cause inflammation of the eye, section anterior to it was usually followed by destructive panophthalmitis. The latest experimenter in this field, Gaule (*Centralblatt für Physiologie*, 1892, p. 419) confirms the statements of Magendie, and holds that only lesion of the Gasserian ganglion itself, or section of the ophthalmic division, is effectual in inducing trophic disorders. These, he holds, occur immediately on section of the nerve, and altogether irrespective of external influences. These trophic disorders consist, according to him, in necrosis of certain portions, with cell proliferations in other portions of the cornea, as well as changes in the corneal corpuscles, Descemet's membrane and aqueous humour, which necrotic and correlated changes, in consequence of external influences, such as drying, mechanical injuries, septic infection, &c., advance to the well-known destructive neuro-paralytic ophthalmia.

---

[1] *Beiträge* bd. x., "Phys. d. Nerv. Trigem."

Our experiments have included section of the so-called ascending root in its course above and through the tubercle of Rolando; section of the trunk of the nerve between the ganglion and the pons; and section of the ophthalmic division in front of the Gasserian ganglion. No special precautions were taken to protect the eye on the side of lesion, which was simply left under the same conditions as the normal one.

Of twelve experiments in which the nerve was divided behind the ganglion, the duration of life was from two days to four months. With the exception of two, which deserve separate mention, none of these cases exhibited any progressive ulceration of the cornea or destructive panophthalmitis. Of those that exhibited any alteration, there was, at most, some degree of conjunctivitis, and haziness of the cornea, as if from drying of the surface. In the majority the cornea remained quite clear and transparent. In one of the exceptional cases above alluded to, in which the eye remained in a perfectly normal condition for eight days after the operation, a drop of collodion accidentally entered the anæsthetic eye. This occasioned intense conjunctivitis, œdema of the eyelids, and a milky white opacity, which overspread the whole of the cornea. Mr. Gunn, who examined this case with us, attributed the opacity of the cornea to the shedding of the superficial epithelium, similar to that produced in man by an irritant, such as lime. The opacity, however, disappeared in the course of a fortnight, and the cornea regained its transparency with the exception of a minute ulcer surrounded by slight opacity, which remained till the death of the animal, six weeks after the operation.

These cases show, therefore, that there is no necessary tendency to progressive inflammation of the eyeball after division of the fifth nerve, behind the Gasserian ganglion, and that such changes as occur do not differ in their process of repair from those that occur in a perfectly normal eye. In one case, after division of the trunk of the nerve, the cornea on the second day exhibited a depressed ulcer and some degree of opacity, which, however, cleared somewhat at the end of a week, but again set in accompanied by a purulent discharge, which resulted in complete disorganization of the eyeball in the course of six weeks. In this case, however, there were indications that the wound was not strictly aseptic, and the temperature remained throughout higher than normal. The probability is, therefore, that the progressive destructive inflammation of the eye was due to septic irritation of the trunk of the nerve.

The most instructive and interesting of our experiments are those relating to division of the fifth, between the Gasserian ganglion and the eyeball. This is the lesion which most observers have found to be specially calculated to excite inflammatory changes in the eye, apparently independent of external influences. We have divided the ophthalmic division in front of the Gasserian ganglion in five cases. In three of these animals, which lived respectively two days, fourteen days, and six weeks, the cornea remained perfectly clear and transparent during the whole time of the animals' survival. Microscopical examination, which was carried out by Dr. Bulloch, showed that the epithelium, corneal corpuscles, and substance of the cornea presented a perfectly normal appearance. In the one which lived six weeks, the anæsthetic cornea did not take on the chloride of gold stain like the normal, but as regards other re-agents there was no change. Occasionally the cornea, when examined by a magnifying glass, was seen to exhibit minute, pin-pointed depressions, such as described by Gaule and Eckhard, as if from partial shedding of the superficial epithelium, but similar depressions not unfrequently occurred also in the sound eye, and they did not appear to have any pathological significance.

In a fourth case the cornea, which had remained perfectly clear and transparent until the fourth day, was irritated by a minute speck of nitrate of silver, and exactly the same condition was established also in the normal eye. This gave rise in each eye to a superficial ulcer with mucopurulent conjunctivitis, but there was no progressive ulceration of the cornea in either case, and the process of repair occurred in the anæsthetic eye as well as in the normal one. In a fifth case, however, the cornea, which had remained clear for two days after the operation, exhibited at the end of that time a central depression with opacity, which progressed until it overspread the whole surface. In this case, however, there were indications of septic irritation in the wound with febrile disturbance.

It results, therefore, from these experiments, that the separation of the eye from the Gasserian ganglion by division of the ophthalmic branch of the fifth, does not necessarily induce any trophic disturbance in the eyeball, or interfere with the processes of healthy nutrition and repair. The only strictly trophic influence of the Gasserian ganglion is, I conceive, on the cellulipetal processes of the Gasserian ganglion cells and their annexes, which probably undergo atrophy, though we have not as yet been able to demonstrate this microscopically. There seems little doubt that the so-called trophic disturbances, which so commonly occur after division of the ophthalmic branch, are due to inflammatory irritation of the nerve, and are of the same character as those which occur in connection with similar irritation of sensory nerves generally. By this irritation, changes are indirectly excited in the corneal tissues, which, under the influence of external agencies and septic infection, result in general panophthalmitis. If, however, proper measures are taken to avoid setting up inflammatory irritation, there is no reason, so far as I can see, why extirpation of the Gasserian ganglion, or section of the ophthalmic division, should excite inflammation of the eye any more than that section of any sensory nerve should be followed by so-called trophic disorders in the region of its distribution, which we know practically is neither a necessary nor, indeed, a common occurrence.

ON HÆMATOMA OF THE DURA MATER ASSOCIATED WITH SCURVY IN CHILDREN.

BY G. A. SUTHERLAND, M.D.EDIN., M.R.C.P.LOND.

Physician to the North London Consumption Hospital; Registrar and Clinical Assistant, Paddington Green Children's Hospital.

THE manifestations of scurvy in children present some differences from those seen in the case of adults. The most important of these are the absence of sponginess of the gums, and the presence of extensive sub-periosteal hæmorrhages in connection with the long bones. The explanation of these variations is to be found, I believe, in the fact that rickets is almost always present, and leads to considerable alterations in the vascular supply of those parts in which scorbutic changes are most marked. In rachitic infants the growth of the teeth is at a standstill, due probably to a diminished blood supply, and sponginess of the gums may not appear during an attack of scurvy. As regards the bones, in rickets there is a greatly increased vascular supply, as shown by the marked periosteal activity, and the thickening around the epiphyses. If scurvy supervene, the hæmorrhagic tendency will naturally show itself in those places where the vascular supply is greatest, and consequently periosteal and epiphysial hæmorrhage is a prominent sign. For a similar reason the active changes present in the skull in rickets seem likely to lead to hæmorrhage in that region if scurvy is present. The occurrence of hæmorrhage under the pericranium has been noted by several observers. With the exception of a doubtful case of Möller's, quoted by Dr. Barlow, I have not met with any recorded case of infantile scurvy in which intracranial hæmorrhage was present. My own experience is limited to the following two cases which were admitted into Paddington Green Children's Hospital. I have to express my indebtedness to Dr. Leslie Ogilvie for permission to publish these notes, and to Dr. Leonard Guthrie for his reports of the *post-mortem* examinations.

Case 1.—*Female child, aged two years: anæmia; fracture of humerus and femur; periosteal effusion; bruise-like extravasations; death; autopsy; hæmorrhage into the brain and its membranes.*

This patient was admitted to hospital on March 21. The child had been healthy when born, and was fed on breast milk entirely for four months. Since that time her food had consisted almost entirely of Nestlé's milk. There had been no snuffles or rash observed. Four months previously she had been put out to nurse. The nurse stated that in the beginning of February the child seemed to have great pain in the right thigh, which was swollen, and she took her to a hospital where disease of the hip joint was diagnosed. Two days before admission the mother got the child back, and noticed a swelling along the right thigh, and also along the left upper arm. There was also a bruise-like discolouration over the right side of the forehead.

The patient was an only child. The father and mother were healthy, and there had been no miscarriage.

On admission the child was found to be fairly well nourished, and had a pale waxy complexion. There were some dirty looking sores along the upper lip, and alæ nasi, and also on the buttocks. A large bruise-like mark was present on the right frontal eminence, greenish in colour. The signs of rickets were well-marked. The anterior fontanelle was open, tense and pulsating. The ribs were beaded, the epiphyses of the long bones were enlarged, and the bones of the forearm were curved. On the left upper arm a smooth, firm, tender swelling occupied rather more than the middle third. The swelling felt like periosteal thickening. The right thigh was bent at an angle a little above the centre, and the femur was fractured. The upper fragment was directed forwards and the lower backwards. The lower part of the shaft was much thickened in its whole extent. No crepitus was elicited between the fragments, but there was extreme tenderness over the whole thigh, and the child cried with a shrill, piercing scream when the limb was touched or moved. The temperature was 96° and the pulse 100 per minute, small and weak. There was nothing abnormal in the heart, lungs or abdomen.

On *March* 23 (two days after admission) signs of cerebral affection were present. The child was sick after food or medicine. Pulsation had ceased in the fontanelle, and the pulse was weaker. There was rigidity of the neck. The pupils were dilated, and the head and eyes were directed strongly to the left side. The left side of the face was drawn up. Both arms were rigidly flexed, and the hands were supinated; there was a good deal of tremulous movement in the arms at times. The child sank gradually, and died the same day.

Necropsy.—The body was fairly-well nourished. A subcutaneous hæmorrhage was present over the right frontal region, and another over the front of the right thigh. The skull was markedly rachitic. On opening the dura mater there was found a large amount of blood, dark and fluid, covering the vertex on both sides, from the middle of the frontal to the posterior part of the parietal regions. The surface of the brain was covered with a layer of blood clot, and the cortex was deeply stained. The blood also extended between the hemispheres. The structures at the base of the brain were covered by a thick, yellowish membrane, which was very tough, and contained no tubercles or nodules. It extended about half-way upon each side, and passed into the Sylvian fissures.

The brain contained two hæmorrhagic cavities under the cortex, situated in the posterior frontal region, one on each side. That on the right side was the size of a small Tangerine orange, and contained grumous, sanious fluid. The cavity on the left side was smaller and more irregular, and was partly filled with a small quantity of yellow, turbid fluid. Under the microscope, this fluid was seen to consist of blood-cells, mixed with a considerable number of fattily degenerated corpuscles. The lateral ventricles contained from two to three ounces of clear fluid. The extravasation under the dura mater seemed to be of some standing, from the considerable amount of organisation present in the membrane covering the base of the brain. The arteries at the base of the brain seemed thickened. On microscopic examination, the the basilar artery was found to be occupied by a thrombus which had undergone some degree of organisation.

The right femur was broken across almost transversely immediately above the centre. There was no attempt at union of the fragments, which were considerably separated. A large amount of soft material was lying round the shaft, under the periosteum, for the most part unorganised, but of a cartilaginous consistence in parts. A similar condition was present in the left humerus, there being no appearance of union between the fragments, and a large quantity of soft material surrounded the broken ends of the shaft. The liver was tough, rather wrinkled and small. The other organs presented no abnormal appearances.

Case 2.—*Female child, aged* 14 *months : rickets ; œdema of eyelids ; anæmia ; cachexia ; hæmorrhages in skin, scalp, and conjunctiva ; ulceration round teeth ; tenderness and œdema of extremities ; syncopal attacks ; death ; autopsy ; hæmatoma of dura mater.*

This patient was seen for the first time on November 11, when she was ten months old. She had had a fit five days previously, which had left her with convergent strabismus. Abdominal pain seemed to be present. The nose and hands were blue, and the mother stated that the child was subject to cold extremities. She was wasted and seemed to have been neglected. The skull was large, and there was distinct bossing in the frontal and parietal regions. The anterior fontanelle was widely open, and the sutures had not closed. The heart was normal, and a good deal of bronchitis existed in the lungs. The abdomen was prominent, and both liver and spleen were enlarged. There was no history of hæmaturia. The patient was suffering from "snuffles," but there was no other evidence of syphilis, nor could any history of it be obtained. Her diet at the time consisted of cow's milk.

As regards the family history, both parents were healthy. There had been twelve children in the family, of whom only three survived, the others having died young, one from jaundice, and the rest from bronchitis. There had been no miscarriages.

The patient had always been subject to bronchitis. She never had fits with the exception of the one mentioned. In the end of the following January the child was seen again. Both upper eyelids were œdematous. She was suffering from another attack of bronchitis, and looked ill. She was admitted to hospital on February 10.

On admission, the patient was moderately well nourished, but markedly anæmic, with a yellow translucent skin. Over the right parietal bone, and part of the occipital, was a soft fluctuating swelling, considered hæmatomatous. The head was large and natiform, with distinct bosses. The anterior fontanelle was open, and the wrists were rather thickened. There were two subcutaneous hæmorrhages on the back about the size of a shilling piece, and another on the inner surface of the right arm. There was no œdema of the extremities. The right arm was kept rigid, and the thumb was firmly adducted; there was also some tendency to adduction of the left thumb. The tongue was furred and dry; and there was some ulceration round the teeth. The liver extended one inch, and the spleen one-and-a-half inches, below the costal margin. The heart was normal, and there was a good deal of bronchitis. The patient was unable to swallow, and had to be fed with the stomach tube. The temperature was 100°, and the pulse 120, rather weak. The urine contained a trace of albumin, but no blood.

On February 12 she had a syncopal attack: the extremities became blue, the pulse could not be felt, and the pupils were contracted. She recovered under stimulation.

On February 15 she seemed to be improving. Her colour was not so yellow. She was still very apathetic, although not unconscious, and cried out whenever she was moved. As a rule she laid perfectly still, moaning at times as if in pain. The thighs were drawn up and adducted.

On March 7 an eruption of hæmorrhagic spots appeared on the abdomen and the back, for the most part small in size. This was accompanied by a rise in temperature, which had been normal for some days, to 101.4°, and by diarrhœa, the motions being green and offensive.

There was another syncopal attack on March 9. On the 13th there were numerous fresh petechial spots over the trunk. The hands and feet were swollen, and the lower limbs seemed very tender to touch. There was a marked increase in the diarrhœa, which had been more or less present during the previous week.

She died on March 26. The child had been lying in a state of torpor since the last note. At times there was a condition of general tonic spasm of the muscles, the eyes being turned to the right. The extremities were blue and œdematous, and the cardiac action was very feeble. The abdomen was extremely retracted. The temperature had been gradually failing, with occasional pyrexial attacks, and for two days before her death it was 95°. No treatment had any effect. The diet consisted of fresh cow's milk, milk puddings, lime juice, and brandy, and medicinally she had grey powder, mercurial inunction and cod liver oil.

Necropsy.—The body was extremely emaciated, with a complete absence of subcutaneous fat. A subconjunctival hæmorrhage was present in the inner and upper surface of the right eye. The teeth and gums were covered with sordes. On the mucous membrane of the lower lip opposite the central incisors, was an ulcer, deeply excavated, with hard, raised edges, and a yellowish base.

The skin was wrinkled and parchment-like. The anterior surface of the body, from the nipple line to the umbilicus was densely spotted with purple, *ante-mortem* petechiæ, mostly the size of a pin's head. There were a few *ante-mortem* bruise-like marks about the extremities, of larger size than those on the trunk, notably one on the outer side of the right leg, and two symmetrically placed on the back of each forearm, the size of a threepenny-piece.

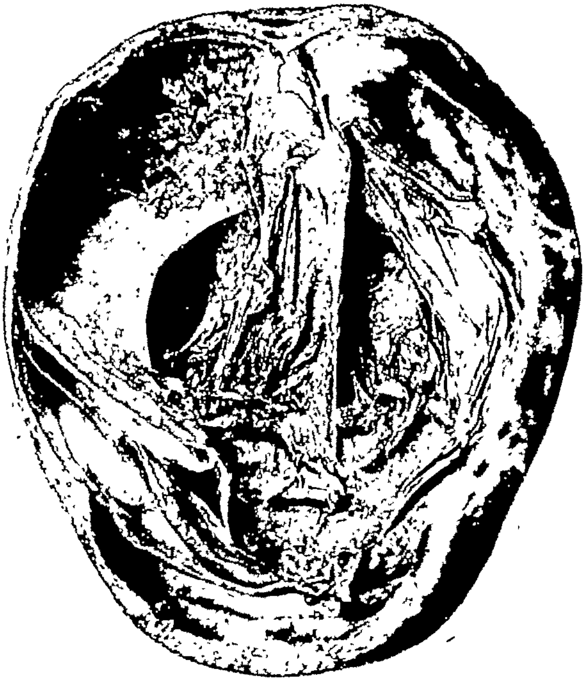
There was a considerable anasarca of both feet and ankles, and clear fluid escaped on puncturing these regions. A more solid œdema occupied the backs of both hands. There were no ulcers or excoriations about the arms or buttocks.

The skull was typically natiform. The parietal and frontal eminences were very protuberant, and the fontanelle was open and depressed. The epiphyses of the long bones were also somewhat enlarged. Incisions made over the tibiæ showed the bones to be normal. There were no periosteal thickenings, and no subperiosteal hæmorrhages. There was no epiphysitis anywhere in a more advanced condition than that mentioned above.

There was some collapse in both lungs, and pleuritic adhesions were present on the right side. The liver was small, mottled on cut section, and there seemed to be some increase of Glisson's capsule along the portal vessels. The spleen, kidneys and intestines appeared normal.

On dissecting off the scalp, a ragged red appearance was noted on the inner surface covering the right parietal bone. A large hæmatoma had been situated here three weeks before death. The parietal and frontal eminences, and also the squamous portions of the temporal bones, were much thickened, of a deep red colour, and soft enough to be indented easily by the finger-nail.

Beneath the dura mater there was in the situation of the subdural space, a dense, fleshy, purplish red, smooth and soft deposit, which completely covered the surface of the brain. It was generally loosely adherent to the under surface of the dura mater, and could be easily peeled off, except in the neighbourhood of the sinuses, where it was more firmly attached. The dura mater above it was white and glistening. The deposit varied from one-sixteenth to nearly three-quarters of an inch in thickness, the thickest parts being towards the base of the skull in the anterior and posterior fossæ. Beneath this layer, and easily



Case 2.—Vertex of skull shewing false membranes *in situ*. The different layers, with cotton wool pledgets interposed, are seen on the left side of the skull anteriorly.

separable from it, was a second dense white membrane, and on the under surface of this was a third layer of soft, yellowish, coagulated lymph, somewhat closely adherent to it, and to the pia mater beneath. On removing this third covering, the brain was exposed, and presented the following unusual appearances. It was generally atrophied, and weighed fifteen ounces. The convolutions were ill-defined, and narrow, and the sulci were obscured, particularly the fissures of Sylvius and Rolando, by a layer of yellowish solid lymph, encircling the contracted and anæmic blood-vessels, and causing the pia mater to bulge externally.

The convolutions generally were of a pale greyish colour. In striking contrast to these were certain limited convolutions, which were of an entirely different aspect. The latter were of a dirty orange yellow colour, considerably depressed below the general surface of the convolutions, feeling so hard and dry to the touch that they were thought at first to be the seat of old caseating gummata. However, on section, it seemed that only the pia mater and the extreme outer surface of the grey matter were thus affected. The pia mater was thickened and gritty, and perhaps the yellow appearance was due to a fatty degeneration of old inflammatory deposits.

The various cranial nerves were white and glistening, and to all appearance normal. There was no thrombosis of the sinuses, and no disease of the petrous, or any other bone. On section, the brain was found to be tough, but otherwise normal.

On opening the spinal canal, there was a deposit of soft material like red currant jelly beneath the dura mater, and covering the cord, particularly well-marked in the posterior dorsal region. It seemed justifiable to regard this as an effusion of blood-stained lymph, and as perhaps resembling the early stage of the much more advanced effusion in the cranial subdural space.

Microscopic Examination.—The false membranes around the brain presented the appearance of blood clot in different stages of organization. In some parts they consisted of fibrinous tissue containing unchanged blood-cells, in others organization was more advanced, and strands of connective tissue were found, forming spaces which contained groups of altered blood-cells.

In the liver there was a slight increase in the amount of connective tissue surrounding the portal vessels, but the hepatic cells were unaltered.

Remarks on Case 2.—In this case we have a combination of diseases. The signs of rickets and scurvy are, I think, unmistakable, and there is also a certain amount of evidence in favour of syphilis. The distinct bossing of the skull is by some regarded as distinctive of syphilis, while by others, among whom is to be included Dr. Barlow, it is accounted a manifestation of rickets. The question has not been definitely settled, and is not of special importance in this case, for while the possibility of syphilis being present may be admitted, the whole series of symptoms, and the pathological changes found at the *post-mortem* were not such as are present in syphilitic disease, but were quite in accordance with a diagnosis of rickets and scurvy. Further, mercurial treatment had been employed without benefit, both while she was attending as an out-patient, and after her admission to hospital.

Remarks on Cases 1 *and* 2.—In both of these a leading feature was the condition known as pachymeningitis interna hæmorrhagica, or hæmatoma of the dura mater. This was extremely well marked in the latter case, and was present to a slighter extent in the former.

The best account of this affection is given by Huguenin in Ziemssen's "Cyclopædia of the Practice of Medicine" (vol. xii.) He says: "Pachymeningitis occurs in scurvy; here, however, we have no personal observations to offer. Many cases exist where syphilis is supposed to be the original disease; the connection is not clear; certainly no one will be disposed to refer the affection to Heubner's disease of the cerebral vessels."

In Dr. Immerman's article on scurvy in Ziemssen's Cyclopædia (vol. xvii.), which is extremely full and comprehensive, the occurrence of intracranial hæmorrhage is regarded as very exceptional amongst adults. He says: "As very rare local affections of scurvy are to be considered the isolated cases of intermeningeal hæmorrhage, which usually run their course under the form of rapidly-fatal apoplexy, but also in some cases lead to death with the appearance of gradually augmenting compression of the brain, headache, dulness, and finally stupor ; it is still doubtful whether or not hæmorrhages occur in the substance of the brain." He does not record any case with *post-mortem* examination.

Huguenin refers to Virchow's theory that pachymeningitis is a hæmorrhagic inflammation of the dura mater, but is of opinion that inflammation is never present, and that the whole process consists of extravasation of blood, at first from the dura mater, and subsequently from already existing false membranes.[1] As regards the pia mater, he has frequently seen " the changes attributed to recurring fluxionary hyperæmia (formation of small fibrous callosities, or at times a thick, dense, leathery membrane, in connection with atrophy of the brain)." He has also observed " a yellow colouration of the arachnoid, of the subarachnoid fluid, and even of the surface of the brain, penetrating to a greater or less depth." Amongst all the cases that Huguenin was able to collect, he found that 2·7 per cent. occurred in children under twelve months, and 2·7 per cent. in children from one to ten years of age.

These appearances recorded by Huguenin are very similar to the ones I have described in the two cases under consideration. About the nature of the meningeal changes there can be no doubt ; the only question is as to the primary disease, for hæmatoma of the dura mater is always a secondary affection. In the history of the cases I have adduced the evidence on which I base the diagnosis of scurvy. In hæmatoma of the dura mater we find :

(1) A tendency to recurrent attacks of hæmorrhage without local inflammation.

(2) That coagulation and organization of the extravasated blood occur, and

(3) That the effects are entirely those of mechanical pressure.

These conditions are also characteristic of the subperiosteal and other hæmorrhages of scurvy.

In Case 1 there was no evidence during life as to the

---

[1] Dr. Wigglesworth also supports the non-inflammatory view. *Vide* BRAIN. Parts lix. and lx., 1892. " Remarks on the Pathology of so-called Pachymengitis Interna Hæmorrhagica."

OBSERVATIONS ON A CHINESE BRAIN.

BY C. HUBERT BOND, B.Sc., M.B., C.M.Edin.,
Pathologist and Assistant Medical Officer, London County Asylum, Banstead.

THE following is a description of the external anatomy of a Chinese brain, including notes as to its weight and capacity. It belonged to a Chinaman, aged 36, who was admitted into this asylum last year suffering from general paralysis. Of this the physical signs were the most pronounced. His gait was very weak and unsteady, tongue markedly tremulous, speech very defective, almost abolished, pupils irregular and unequal, knee reflex exaggerated. Mentally he manifested a considerable amount of *bien être*, laughed in a silly, fatuous manner, and at times had many delusions of grandeur. The disease was cut short by the patient dying seven weeks after admission from an attack of pneumonia, and we may fairly take it that there had not been sufficient time for it to produce any marked atrophy of the convolutions which might cause an apparent alteration in their arrangement.

Literature.—The first report on any Chinese brain appears to be that[1] in 1886 by Mills and Parker. Three others[2] were described soon after by Benedict. Dercum in 1889 reported two more,[3] and again in 1892 he published notes on an additional one.[4] As far as possible a comparison will be made between this eighth Chinese brain now under consideration, and the above previously described ones.

---

[1] "Preliminary Study of a Chinese Brain," by A. J. Parker and C. K. Mills, *Journal of Nervous and Mental Disease*, vol. xiii, No. 10, 1886.

[2] "Drei Chinesen Gehirne," by Prof. Dr. M. Benedikt, *Medicinische Jahrbucher*, 1887.

[3] "A Description of Two Chinese Brains," by F. X. Dercum, *Journal of Nervous and Mental Disease*, July, 1889.

[4] "Note on a Chinese Brain," by F. X. Dercum, *Journal of Nervous and Mental Disease*, September, 1892.

Shape and Size.

Without resorting to the balance, the brain is easily noticed to be considerably smaller than those we are accustomed to see, and to be of a peculiar shape. The difference in shape appears chiefly due to the increase in the greatest transverse diameter as compared with the antero-posterior. On actual measurements of the cranial cavity, the greatest width was found to be 14 cm. (5·5 in.), and the extreme length 15·5 cm. (6·125 in.). The cephalic index is therefore 90, and the skull a markedly brachycephalic one. The hemispheres viewed from above do not display their usual fairly regular oval contour. From the region of the parietal eminences, the point where they attain their greatest width, they rapidly taper towards the extremities, reminding one of the simian cerebrum; this is chiefly noticeable posteriorly, causing the occipital lobes to look small and pointed. From the hinder part of the corpus callosum the hemispheres appear to gradually gape asunder till at the extremities of the occipital lobes they are an inch and a half apart. Looking at the base of the brain, there is none of the eversion of the basal surface of the frontal lobe, noted as being present in all the previously described specimens, but there is well marked eversion of the basi-temporal surface. Benedict noted this in each of his three specimens. In this brain the anterior parts of the temporo-sphenoidal lobes are poorly developed, being thin and wedge-shaped.

The weight of the whole brain is 1,182 grammes (38 oz.), that is, 176 grammes less than the weight of an average male adult brain. This diminution in weight is not shared by each division of the brain, but belongs to the cerebral hemispheres; for these weigh together only 995 grammes, while the average weight of them in a normal European brain is 1,171 grammes. The cerebellum weighs 163·2 grammes, and the pons and medulla together 23·3 grammes, these being just about average weights.

The cubic capacities of the several parts were ascertained by immersing them in graduated glass measures, partially filled with water, and the following figures were obtained :—

Capacity of the hemispheres, 822 cc. (50 c. in.); cerebellum, 141·7 cc.; pons and medulla, 21 cc. Comparing, in man and in apes, the proportion the cerebral hemispheres bear to the cerebellum, in the case of the former it is as 8½ to 1 and 5¾ to 1 in the chimpanzee (Huxley). Making a similar calculation in this Chinese brain the proportion is exactly 5 to 1. Supposing that our specimen is fairly typical of a normal brain of that race, and even allowing that there was sufficient time for the disease from which the man was suffering to produce some atrophy, even then there must be a considerable difference in the degree of the relative development of the cerebrum and of the cerebellum to account for the above remarkable change in the proportion.

THE FIVE FISSURES.

The Fissure of Sylvius gives off quite normally on each side its small anterior and ascending branches, and continues backwards as usual as the posterior branch. In two of the previous seven brains it is mentioned as being unduly long. In this specimen, as far as the spot where it turns upwards into the tail, it is of normal length; but the tail itself on both sides can be distinctly traced further than usual, especially on the left side. This point will again be referred to under the parietal lobe. On both sides the tail, instead of inclining upwards and backwards, practically forms a right angle with the posterior branch of the fissure, and thus comes really to be directed more forwards than backwards.

The Parieto-Occipital Fissure is well marked on both sides. As regards that part of it on the mesial surface of the hemisphere (the internal parieto-occipital fissure), there is nothing special to notice. Benedict noted that in one of his specimens it did not become on one side confluent with the calcarine fissure. In this brain there is no such abnormality on either side; the calcarine fissure is reached in the usual position, namely, about its middle, on a level with the posterior extremity of the corpus callosum. That portion of the fissure which is situated on the dorsum of the hemisphere (the external parieto-occipital fissure) is interesting in regard to its length. It makes its appearance at the normal point, midway between the sulcus of Rolando and the occipital extremity of the hemisphere, but is carried to a remarkable extent outwards on both sides, particularly on the right. This part measures generally much less than an inch. In the specimen in hand it extends outwards for an inch and a half on the right side, and an inch on the left. If we take into consideration the general small size of the hemispheres, the length of these fissures would be comparatively greater still. Quain states that the size of the external parieto-occipital fissure depends inversely on the size of the convolution which curves round its outer extremity and connects the parietal with the occipital lobe. And he goes on to say that in consequence of the development in man of this and similar connecting convolutions, this fissure is much less marked in the human brain than in that of the higher apes.

The Calcarine and Hippocampal (or Dentate) Fissures.—In our specimen these two fissures follow a perfectly normal course, except that the former departs somewhat from its usually fairly horizontal direction, its most posterior part being markedly sinuous. On neither side is there the slightest attempt at any confluence between them, the gyrus fornicatus being well developed throughout its whole extent. In each of the three brains described by Dercum, this union took place on both sides; it was present also on both sides in one of Benedict's three cases, and Mills and Parker in their specimen noted that on the right side the gyrus fornicatus became greatly reduced at this spot, the two fissures very nearly becoming continuous.

The Collateral Fissure (Occipito-Temporal) is well marked on both sides, and shows no departure from the normal.

THE FRONTAL LOBE.

The Furrow of Rolando (Central Sulcus) belongs, of course, as much to the parietal as to the frontal lobe, but it is convenient to consider it now. In the previously described Chinese brains the point, on which stress is laid with regard to it, is the presence or absence of union between it and the Sylvian fissure. In nearly every instance on both sides was this confluence complete or almost complete. It was very nearly so on both sides of the Mills-Parker brain, on the right side of Benedict's first reported specimen, in both hemispheres of his second, on the left side of his third, on the right side of Dercum's first brain, on both sides of his second, and in the left hemisphere of his most recent case. It was freely confluent with the fissure of Sylvius on the right side of Benedict's third specimen, on the left side of Dercum's first, and in the right hemisphere of his latest brain. This apparently regular confluence or approach to it is on neither side observable in the specimen in hand, and certainly on the right side there is a wider loop of grey matter than usual connecting the ascending frontal and parietal convolutions and shutting off the sulcus of Rolando from the Sylvian fissure. The sulcus at its upper end notches the upper margin of both hemispheres, but on neither side is it continued on to their mesial surfaces. Curiously the upper extremity of the right Rolandic furrow is nearly a quarter of an inch behind its fellow; this is seemingly accounted for by the greater length of the superior frontal convolution on the right side. In both hemispheres the sulcus pursues an uninterrupted course.

The Præcentral Sulcus (*Transverse Frontal Furrow*) is not nearly so much broken up as is usual, and is almost as prominent as the central sulcus, and, as we might therefore expect, there is a particularly well developed ascending frontal (anterior central) convolution. The sulcus on neither side is continued completely to the upper edge of the hemisphere, nor is it on either side confluent with the Sylvian fissure, and therefore, at any rate in this case, cannot be regarded as the prolongation of the ascending branch of that fissure.

The Superior, Middle, and Inferior Convolutions are well developed and distinctly mapped out on either side by the two horizontal sulci. They are subdivided by other secondary

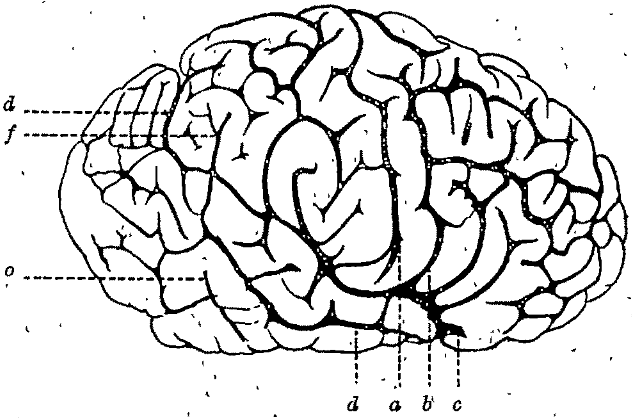


FIG. 1.—Exterior surface of right hemisphere.

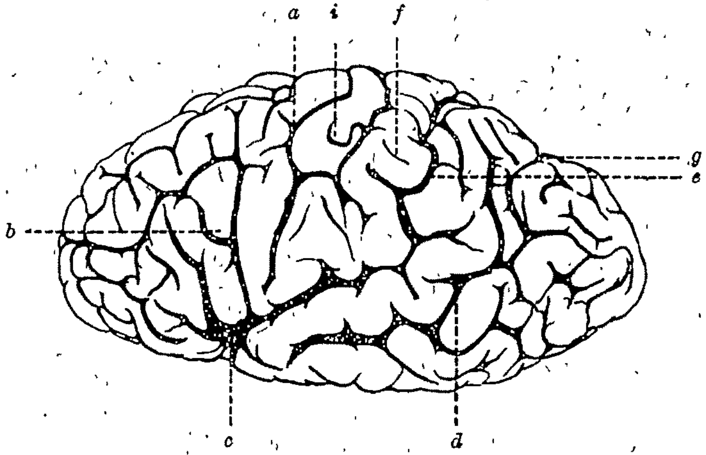


FIG. 2.—Exterior surface of left hemisphere.

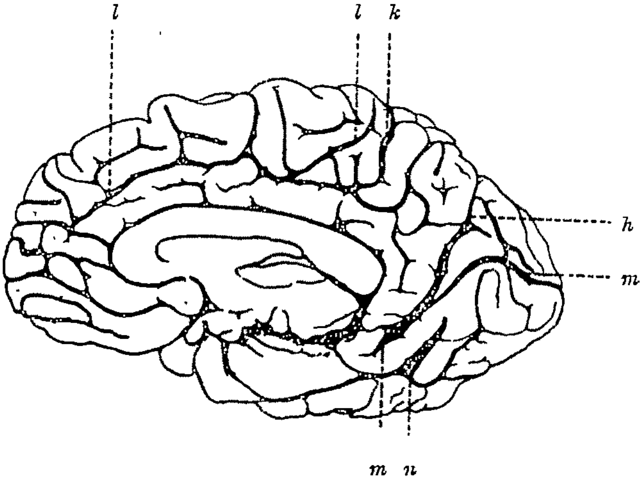


FIG. 3.—Mesial surface of right hemisphere.

a. The sulcus of Rolando.
b. The præcentral sulcus.
c. The Sylvian fissure.
d. The parallel sulcus.
e. Shewing the marked prolongation of the Sylvian fissure on the left side.
f. The external parieto-occipital fissure.
g. Position of the left parieto-occipital fissure.
h. The internal parieto-occipital fissure.
i. The ascending parietal convolution.
j. The provisionally termed posterior ascending parietal convolution.
k. The continuation on to the mesial surface of the post-central sulcus.
l. The calloso-marginal sulcus.
m. The calcarine fissure.
n. The collateral fissure.
o. The middle temporo-sphenoidal convolution.

NOTE.—The upper limit of fig. 2 does not correspond with the upper edge of the hemisphere, but to a line about an inch external to it; hence the marked transverse furrowing is not so well shewn as it might be and the parieto-occipital fissure is not shewn at all.

sulci. The greatest complexity is in the fore part of the frontal lobes; here the gyri are considerably multiplied, and look small and closely packed.

The Orbital Lobule.—The absence of any eversion of this part of the frontal lobe, noted as being present in all the other seven Chinese brains, has been already referred to. The mapping out of its surface into interior, anterior and posterior gyri is not easy; the only one well marked is the internal, and in it on either side is a deep olfactory sulcus.

The Parietal Lobe.

The Intraparietal and Postcentral (Retrocentral) Sulci will be more conveniently studied together. According to the usual description, the former is said to ascend parallel to the sulcus of Rolando for half its extent, and is then directed backwards, blending occasionally with the superior occipital sulcus; and its original direction upward is said to be continued as the postcentral sulcus. On examining the specimen in hand, however, such a description is at once seen to be inapplicable. Here in both hemispheres parallel to the furrow of Rolando is a well marked sulcus, almost as pronounced as the central sulcus itself, in fact more so at its upper end, as it is continued well on to the inner aspect of the hemispheres. On neither side does it open into the fissure of Sylvius. No attempt whatever at any branching from this large transverse furrow is seen on the left side, but in the right hemisphere at the spot where the intraparietal sulcus usually changes its direction, a small branch is given off. Thus in this brain the name postcentral sulcus, besides what is usually meant by that term, would more correctly include the ascending part of the intraparietal furrow. As a result there appears to be in both hemispheres an additional ascending parietal convolution, though on the right side it should be noted that this posterior ascending parietal convolution (if one may so term it) is not so distinct as on the left.

Behind this region the sulci mapping out the remainder of the parietal lobes into other gyri are far from symmetrical on both sides. In the left one the tail of the fissure of Sylvius is continued upwards to a remarkable extent, practically forming a new transverse furrow taking a course similar to that generally described as belonging to the intraparietal sulcus, for it can easily be followed to within an inch and a half from the upper margin of the hemisphere, and thence is directed backwards at a right angle past the outer extremity of the parieto-occipital fissure and blends with the superior occipital sulcus. In the right hemisphere the configuration of the parietal lobe is more in accordance with the normal. For, as above stated, there is a branch from the large transverse sulcus behind the furrow of Rolando, perhaps representing the hinder part of the intraparietal sulcus; and the tail of the Sylvian fissure can only be said to be continued upwards slightly more than usual. In the previously described Chinese brains there were no variations in the intraparietal sulcus that we are not frequently in the habit of seeing.

The Superior Parietal Lobule.—As follows from the fact that the postcentral sulcus, we saw, was continued on to the inner surface of the hemisphere, on neither side is the upper lobule connected with the true ascending parietal convolution. But in both hemispheres the first annectant gyrus is plainly traceable joining the superior parietal lobule to the upper occipital convolution. On account of the great length of the external parieto-occipital fissure, this annectant gyrus is very sinuous and thin, particularly so on the right side.

The Inferior Parietal Lobule.—Its anterior part termed the supramarginal gyrus appears on the left side to be represented in the lower part of what above has been provisionally called the posterior ascending parietal convolution. That this is so may be gathered from the name given by Turner to this portion of the lobule, namely, the convolution of the parietal eminence, as this part by its protuberance evidently corresponds to the boss of the parietal bone. On the right side, owing to the less complete development of the so called posterior ascending parietal convolution, the supramarginal gyrus is fairly normal. The angular convolution, that is, the hinder part of the inferior parietal lobule, arching over the upper end of the parallel sulcus, along with the second annectant gyrus, can fairly clearly be traced in both hemispheres.

THE OCCIPITAL LOBE.

In the three specimens of Dercum, both lobes were observed to be distinctly smaller than normal; especially was this seen in the cuneate lobule. As regards the specimen in hand this fact has been already noted when the general shape and size of the brain were being considered. In the left occipital lobe sulci can be traced dividing it into superior, middle, and inferior convolutions. The third and fourth annectant gyri are seen connecting the last two with the middle and inferior temporo-sphenoidal convolutions respectively. But on the right side three occipital convolutions cannot be made out. The lobe seems divided into two nearly equal parts, the upper, as before said, being joined to the superior parietal lobule by the first annectant gyrus, and the lower has an annectant gyrus connecting it with the inferior temporo-sphenoidal convolution. On neither side is any indication of a transverse occipital sulcus, that is, the small groove said to be generally present in most brains, and directed outwards across the upper part of the occipital lobe a little behind the parieto-occipital fissure. When present it is said to represent the outer portion of the external perpendicular fissure of the ape's brain (*vide* Quain).

THE TEMPORO-SPHENOIDAL LOBE.

The Parallel or Superior, Temporo-Sphenoidal Sulcus is an important one to observe on account of its constancy in all Primates. In the four brains reported by Dercum, Mills, and Parker, attention is drawn to its great length and confluence with numerous perpendicular furrows. In the specimen now studied it is well defined on both sides, and follows very accurately the course of the Sylvian fissure, first backwards, and afterwards being directed perpendicularly upwards, at exactly the same level at which the fissure of Sylvius changes its direction. Its continuation upwards is carried to a remarkable degree on both sides. But there are none of the transverse confluences mentioned in previous brains observable here, the only junction being that on the left side at the spot where the parallel sulcus changes its direction, the middle occipital furrow opens into it.

Thus a superior temporo-sphenoidal (inframarginal) convolution is well marked on each side. In the right lobe middle and lower temporo-sphenoidal sulci are fairly defined; the former, however, is broken about its centre by a gyrus connecting the middle and lower convolutions together. The middle convolution has no annectant gyrus joining it to the occipital lobe, as is generally the case, but is quite cut off by a small branch from the parallel sulcus first directed backwards, then descending, and ultimately winding into the middle temporo-sphenoidal furrow. Below the inframarginal convolution the left lobe is very irregularly subdivided into small gyri and middle and inferior sulci cannot properly be defined.

THE MESIAL SURFACES.

The Calloso-Marginal Sulcus.—The usual description given for it applies here in both hemispheres of this specimen, but its termination is not quite symmetrical on the two sides. As is generally the rule, on both sides it ends a short distance behind the upper extremity of the furrow of Rolando, but, while on the right it merely notches the upper margin of the hemisphere, on the left it becomes confluent with the postcentral sulcus. Mills, Parker and Dercum refer to the special complexity of the calloso-marginal sulcus and vegetative repetitions of it were observed by them. None of this is noticed in the present specimen; but in the right hemisphere, as was the case in Dercum's third brain, an apparent addition to the length of the sulcus is seen by its original direction being continued by a furrow in the quadrate lobe also ending by being bent upwards and notching the hemisphere's upper edge.

As regards the convolutions on this surface, no noteworthy peculiarities are observable. The small size of the cuneus has already been referred to, as also has the fact that the gyrus fornicatus is well developed and does not allow of any attempt at confluence between the calcarine and dentate fissures. The dentate convolution (fascia dentata) was not more pronounced than usual; this fact is noted because in some animals it is much more greatly developed than in man.

SUMMARY.

Before summarizing the above facts it may be well to state here that as regards the markings on the island of Reil and the configuration of the cerebellum, pons and medulla, no departure from the normal is observable.

Although several decided differences from what we look upon as the normal have been pointed out in this and the other seven Chinese brains, the material studied has been too limited to justify general conclusions. But there are certain features which occur with such marked persistency, some of them in all, and others at any rate in the majority of these specimens, that they cannot be regarded as mere coincidences.

First of these stands out the greater prominence given to furrows running transversely as compared to those in the antero-posterior direction. This has been pointed out in every specimen, and is well illustrated in the present case by the three large sulci running parallel with each other in both hemispheres, namely, the central itself with the præ- and postcentral sulci practically as distinct as itself. And in addition to these we have seen on each side an unusually long external parieto-occipital fissure, and that the parallel sulcus was prolonged to a remarkable extent in a transverse direction. This pronounced perpendicular furrowing is still further accentuated on the left side by the undue extension upward of the Sylvian fissure.

Then, as to convolutional complexity, taking the brain as a whole, it is certainly up to the normal standard, and in the frontal lobes even rather beyond the average; this latter point was observed, too, in the first pair of Dercum's cases.

In all his three specimens the occipital lobes, and more especially the cuneate lobules, were noted as not attaining the normal size. This has already been mentioned as being the case in the present specimen and thus is a point which is constant in at least half the number of brains studied.

If this brain is at all typical of the race to which it belongs, then the small size and weight of the cerebrum as compared to the cerebellum is a point worthy of special emphasis.

It is hoped that the above observations may prove of interest to those who study the comparative anatomy of the human brain, and that as more material accumulates, generalizations of a greater value may be obtained.

ON GENERAL PARALYSIS

(WITH A CRITICAL DIGEST).

BY W. JULIUS MICKLE, M.D., F.R.C.P. (LONDON).
Grove Hall Asylum, London.

I.

ANATOMY IN RELATION TO GENERAL PARALYSIS OF THE INSANE.

IN order to clear the way for the discussion of the pathological anatomy of general paralysis, it is desirable to speak beforehand of (A) the normal anatomy of the intercellular, or interstitial substance, or stroma, or matrix of the brain, particularly of the brain cortex; and (B), in a subsidiary way, to refer to disputed points concerning the lymph-system of the brain. These are, and are yet to be, battle-grounds of anatomists.

A.—THE INTERSTITIAL, OR INTERCELLULAR, OR CEMENTING SUBSTANCE, OR MATRIX, OR STROMA OF THE BRAIN, OFTEN TERMED NEUROGLIA, BUT PART OF IT, ALONE, RECEIVING THIS NAME FROM SOME.

One must leave aside discussion of the numerous irreconcilable observations and views on this tissue or substance. It has been termed a non-nervous constituent of the brain; has by most (as *e.g.*, Witkowski, Ziegler, Obersteiner), been placed among the connective tissue structures, and has been variously divided. Nevertheless, Robin and Loewe long ago declared it to be nervous matter of a simple kind; and Klebs expressed a similar opinion in speaking of pathological cases. As Gierke stated, the supporting tissue of the central nervous system is comparable with connective tissue in work or functions generally, but essentially distinguishes itself therefrom by its development, appearance, &c. It has often been described, and usually under the name neuroglia, as being partly of homogeneous, structureless, or, again, finely granular, or molecular, or even fibrillated-looking, material—partly of a structure constituted of fine connective-tissue fibrils, and of cells forming their origin or nodal points—and partly of nuclei, or "free nuclei."

Obviously, its functions are not only to form an imbedding or protective material, but also a supporting structure or scaffolding for the delicate and vulnerable anatomical nervous elements, and for the minute blood-vessels. Less obvious, but now sufficiently clear is also the existence of plasmatic functions of portions of it, in relation to the local lymph-circulation, and hence to brain nutrition and welfare. And, to some extent it may have lowly nervous function, irrespective of that of the definite nerve-fibrils coursing in, or through, it. Just as these several functions vary in relative importance and necessity in different parts of the nervous system, so do we find corresponding differences in the relative preponderance of one or another of the several constituents of this tissue and substance. Thus, in the outmost, or superficial, layer of the cerebral grey cortex is a closely-woven feltwork of connective tissue; whilst throughout the cortex generally the so-called "ground-" or glia-substance (presently to be described) appears to be, relatively, in unusual quantity; on the other hand in the white brain-matter the reticulum of glia-cells and fibrils, with little, if any, ground-substance supports the nerve-fibres; and, again, at the radial septa of the spinal cord, and associated with the large densely-ranked nerve fibres, is a close network of neuroglia cells and fibrils, with but little ground-substance.

To clear the present use of the terms employed, I must enumerate, in detail, the several different materials described as constituting this tissue material, which is disposed between the units of the cortical nerve-cell-and-fibre apparatus, and blood-vessels.

(1) The ground-substance, or cement- or glia-substance; to which constituent alone, some (as, recently, Obersteiner, and Binswanger in places) restrict the name neuroglia—

a name occasionally limited (nominally, but not really, if what I state below is correct), to sustentacular tissue of ectodermal origin[1]—and often applied to the whole tissue and material in question, as sometimes, indeed, to the whole, is the name "ground-substance" itself,—incidental proofs of the enormous discrepancies in nomenclature on this subject.

It has been described, variously, as being unformed, structureless, homogeneous and softish; or by others as being molecular, or finely granular:—

(2) Free glia nuclei, or neuroglia nuclei.

(3) Fibrils and processes; and

(4) Cells, of various types (both "(3)" and "(4)" to be described below).

As to the first of these, "(1)," much of that which was termed glia-, or ground-, or cement-substance has been microscopically resolved into medullated and non-medullated nerve-fibres and fibrils; and the fine granules of neuroglia ("interfibrillar granules" of Boll) attached to processes of nerve-cells and connective tissue, have been stated (Obersteiner) to belong to the nervous system and not to connective tissue.

By some recent methods of investigation, the spaces between the nerve-cells are found to be largely occupied by a host of nerve-fibres and fibrils, formed not only by the collaterals and terminals of nerve-cells, but also by those of fibres coming from the corpus callosum and other distant organs, and by the collaterals of association-fibres.

As to the existence of the nuclei forming the second "(2)" of the above-mentioned constituents of brain-stroma, and as to the morbid appearances attending their asserted proliferative hyperplasia; it is probable that these so-called "nuclei" are, or mostly are, cells in reality; and the appearance explained as being due to a supposed nuclear proliferation, as in general paralysis, has been attributed otherwise (as by Lubimoff) to an effusion of lymphoid elements; or of leucocytes. These "nuclei," may, therefore, be provisionally accepted as cells with little cell-protoplasm, even by those who still look upon them as representing hyperplasia of natural elements of the part. They stain highly with carmine, and some aniline colours.

---

[1] As by Ph. Chaslin, *Archives de Méd. Expérimentale,* May, 1891, p. 339.

Of the elements, of the tissue under discussion, enumerated above, there remain, therefore, only the third and fourth, " (3) " and " (4)," as being of paramount importance in either normal or abnormal states; and they will now be dealt with conjointly, *i.e.*, the cells and the processes or fibrils pertaining to them.

To be brief, these cells with their appurtenant processes and fibrils may be divided into two groups.

(*a*) *First group.*—One of them consists of *primary* glia-cells, which are of ectodermal (epiblastic) origin, and under certain aspects or methods of viewing them, have a horny appearance. From some of these proceeds the thick layer of tangential horizontally radiating fibrils, already referred to, in the outermost layer of the cerebral cortex (where also, in parts, are the apical processes of large pyramidal nerve-cells gaining the surface from the deeper cortical layers, as shown by Retzius); whilst the bodies of the glia-cells of this first group also send far down into the cortex, and in rich profusion, long, slender, uniform, unbranched, nearly straight, fragile fibrils; the fibrils from numerous cells form a network by commingling and intercrossing.

At first glance, these cells with their processes have a general aspect slightly like horse-tails. Others of the glia-cells of this group, and found in the white substance of the brain, give off similar fine fibrils, radiating in various directions, so that they have a thistle-down aspect.

As believed by Ramon y Cajal, Binswanger, and others, these cells and their processes are, embryologically, of epithelial source, and independent of the blood-vessels. They are primary glia-cells, or primary cells of the supporting framework or scaffolding of the cortex; and, of the two, are the kind to which the aspect of cornification is limited (Binswanger), although Gierke has attributed this conditional horny look to all glia-cells. The observations of His (1883 to 1890) throw some light on the subject of these cells, as also do those of W. Muller. For example, in the embryological development of the spinal cord, His found two distinct separate epithelial-cell forms; one of which provides the spongioblasts which serve for the construction of an embryonic frame-tissue or stroma; the other is formed of the germ-cells—becoming neuroblasts—from which the nerve-cells and fibres develop; although, from their origin, the glia-cells and nerve-cells have separate conditions of growth increase and further development. Hence the glia-cells of this first group are epiblastic (or ectodermal) in origin, and, like the nerve-cells and fibres, arise originally from embryonic epithelial elements.

They have primarily a supporting function, forming, as they do, a framework or scaffolding for the nervous elements.

(*b*) *Second group.*—Occurring freely in the brain cortex, the second group consists of glia-cells and their processes—cells which in normal states are flask-shaped, or of more or less stellate aspect (Deiter's cells); are of mesodermal (mesoblastic) origin; and, besides some finer processes, have numerous protoplasmic or dendritic processes, most of which are comparatively short, stout, coarse, and one or several of which are seen to be attached to the perivascular sheath or adventitial tunic of adjacent blood-vessels. When morbidly hypertrophied, as in general paralysis, these cells and processes have the bloated succulent appearance, and the conformation, to which it would be well to restrict the name "spider cells."

Probably mesodermal in origin, a point held to be uncertain by Ramon y Cajal, the cells of this group are *secondary* glia-cells; embryologically, are, seemingly, from connective elements which come in with the invading blood-vessels;[1] are intimately associated with those vessels throughout life; seemingly are plasmatic cells or lymph-system elements;[2] provide a lymph-canalicular system between the blood-vessel- and lymph-channels of the cortex of the brain; and, in addition to their well-known connection with the blood-vessels and perivascular (extra-adventitial) lymph-space, and hence indirectly with the epicerebral space, also, it has apparently been made out with likeliness, in recent observations, and as stated by Dr. Andriezen, that their body and coarse processes are surrounded by a system of pericellular lymph-spaces.

---

[1] The well-tinting nuclei observed here in the second half of fœtal life, appearing simultaneously with the ingrowth of blood-vessels, and subsequently increasing greatly in number, are taken to be connective tissue cells by Binswanger

[2] "Lymph-connective elements," or "scavenger cells," of Bevan Lewis.

Obviously, they are not primarily, but only indirectly, sustentacular elements; apparently they are especially plasmatic, *i.e.*, lymphatic, in function, and are alleged to be the scavenger cells of the brain. As compared with the cells of the preceding first group, they exhibit more of vital phenomena, have little power of resistance, are much more vulnerable, and do not put on the horny appearance.

Nevertheless, some have even recently still described the spider cells as being connective tissue cells (*e.g.*, Obersteiner, 1890).

Dr. Alex. Hill suggests that "neurogleia" (as used by another as equivalent to ground-substance) perhaps partly consists of cell-processes not resolved by the microscope, and partly of coalesced substance of such processes, and that it is not necessary to believe in a non-cellular ground-substance. But he also suggests an application of the terms "neuroglia" and "connective tissue," differing somewhat from that used in this communication.

Finally, to be consistent with what has been stated above, one could only partly accept the first, and would reject the second, of views recently expressed (as by Dagonet, p. 413-6 *op. cit.*); for one could only in part admit (1) that the neuroglia (in the wider sense) is an epithelial tissue, and not a connective tissue; and one would deny (2) that spider cells (representing glia-cells of the second group hereinbefore described), are with complete accuracy to be called "epithelial cells of the neuroglia."

B.—The Lymph-System of the Brain.

The statements immediately preceding, as to the relations of the plasmatic glia-cells to the blood-vessels and perivascular lymph-space, to the epicerebral space, and to the formation of a lymph-canalicular system, reveal and explain the vast importance of several conditions and lesions found in general paralysis—namely (with diseased coats of blood-vessels) hyperæmia, even to the dilatation of those vessels, so that sometimes they may even be appressed to the walls of the tunnels which traverse the nervous substance and in which they lie—the hyperplasia of adventitial nuclei, or cellular accumulated heaps in the sub-adventitial and perivascular spaces—the overgrowth, to gross "spider" form, of the second of the groups of glia-cells above described—the adhesions of pia to cortex, occluding parts of the epicerebral space, and closing some of the communications of perivascular lymph-spaces therewith—all of which obviously tend to choke the lymph-circulation of the cortex, and to produce lymph-stasis with all the evils attending it. The importance of lymph-stasis and lymph-circulatory disorder in general paralysis is clearly manifest.

II.

PATHOLOGICAL ANATOMY AND PATHOLOGY OF GENERAL PARALYSIS.[1]

It is unnecessary to reproduce the numerous and differing pathological views maintained concerning general paralysis. One need only recall the important part frequently assigned to active cerebral congestion or hyperæmia; by some taken as the proximate determining cause of general paralysis, by others as being an important but secondary phenomenon, by still others as comparatively unimportant; and recall also the conflict of opinion as to whether the essential pathological change in general paralysis is principally and primarily of an inflammatory, or, on the contrary, of a degenerative nature:[2]—as to the former of which, again, opinion was divided on the seat of the inflammation, and its interstitial or parenchymatous character:—and as to the latter of which there was little harmony on the alleged character and course of the degenerative process.

---

[1] Dr. Otto Binswanger. *Die Pathologische Histologie der Grosshirnrinden-Erkrankung bei der allgemeinen progressiven Paralyse mit besonderer Berücksichtigung der acuten und Fruhformen. Jena*, 1893.

Dr. J. Dagonet. *Annales Médico-Psychologiques*, Nov. 9, 1893. Extract from a Treatise on "Mental Diseases" in the Press by Dr. H. Dagonet.

[2] See the writer's "General Paralysis," second edition, pp. 333-4, 333.

Some use of Dagonet's *résumé* has been made in part of what immediately follows.

Most of the earlier observers of general paralysis took it to be a chronic meningitis, or chronic meningo-encephalitis or peri-encephalitis; conditions of which the cerebro-meningeal adhesions afforded notable indications, although the frequency of these was far under-estimated.

Then, the atrophy of brain following (as supposed) the obvious meningeal changes, led to the statement that general paralysis was a precocious senile atrophy of the brain, following chronic meningitis (Duchek).

Rokitansky led the way in attributing an important part to hypertrophic changes of the interstitial tissue in general paralysis; the hypertrophy, or the proliferation of which tissue, occurring especially in certain parts of the cerebral cortex, he believed to be produced more usually by hyperæmia, but sometimes by an encephalic inflammation proceeding with the meningitis; and he maintained that this increase of connective tissue had the result of disorganizing the brain-cortex, the nerve-fibres and nerve-cells becoming profoundly altered and broken up. By some, a degeneration of the blood-vessel walls was held to occasion [or promote] the disorders of local nutrition and circulation, and the new formation of connective tissue was described around the blood-vessels of brain and pia. By the increase and proliferation of interstitial tissue, leading to destruction of parenchymatous nervous elements, the lesion of general paralysis was held to be brought into line, and to rank, with a cirrhosis of liver or of kidney. And, although he termed them lymph-connective elements, possessing the function of brain-scavengers—a function overwrought in general paralysis, and leading to the well-known overgrowth of the glia-cells of the second group already described, which may then more fitly be named "spider cells"—Bevan Lewis ascribed to them, in this latter state, an aggressive disintegrative effect on the adjoining nervous elements—a view which falls essentially into line with the above cognate view as to primary connective interstitial lesions in general paralysis, and consecutive dependent parenchymatous ones.

Yet in reality the matter was not so simple as a primary interstitial change, and resulting secondary destruction of nervous elements. For the lesions of general paralysis are complexly associated; no one of them, alone, has pathognomonic character; and general paralysis not being always one and the same, these lesions are differently grouped in separate cases; may be unequally distributed, patches of marked change, whether chiefly vascular, or interstitial, or nervous, perhaps appearing beside comparatively normal structures. And of the several individual tissues constituting the brain, the lesions may vary much in intensity between themselves, for marked parenchymatous lesions may accompany slight interstitial or vascular ones, and *vice versâ*; for example, where the nerve-cell degeneration and destruction are extreme, there may occasionally be scarcely any glia-cell overgrowth.

The blood-vessels and the stroma having thus successively been taken as the chief and primary seat of morbid action, the nervous elements in their turn were credited with playing a similar part; and this in recent years especially, although Meschede had long ago declared the pathological basis of general paralysis to be parenchymatous inflammation of the inner layer of the cerebral grey cortex, and principally affecting the ganglionic nerve-*cells*.

Disappearance of medullated nerve-*fibres* in definite local portions of the cerebral cortex was found by Tuczek. This he was inclined to look upon as a primary lesion; for while leptomeningitis and interstitial encephalitis usually co-exist with the nerve-fibre lesion, this last seemed to be independent, at least in some cases. Those which disappear are the finest fibres. This lesion varies in degree in different cases and in different parts of the brain, affects certain portions of it earliest and most, and there is some evidence of a successive order of implication of the several parts affected. Other observers (Zacher, Greppin, Kronthal, Schütz, Wille, Friedmann, Meyer, Lissauer) have considerably modified the observations of Tuczek, whilst confirming them in a general way, and have shown that other parts of the encephalon are also often similarly affected, including the cerebellum and optic thalami, where many nerve-cells and fibres may disappear. And the conclusion gained ground that there is a *primary* process of destruction of these nerve-fibres in general paralysis, to which any sclerosis or neurogliar proliferation found is *secondary* (*e.g.*, Strümpell); although a somewhat different and diffuse primary disappearance of nerve-fibres is also observed in some other diseases of the brain. Moreover, it was asserted by Weigert that in tabes dorsalis the nervous elements similarly first undergo degeneration, that the tabetic sclerosis also found is only secondary thereto, and that the same order of events occurs in all cases of proliferation of neuroglia. In general paralysis, Schütz likewise observed destruction and disappearance of the finest fibres of the grey central ventricular nervous substance, which, from a developmental point of view, are stated to form a system, and are, in likelihood, fibres of association between the different cells of the central nuclei of nerves, and so conduce to the synergy of those nuclei. On that basis, this lesion would be systemic or systematic, and rank analogously to the systematic columnar lesions of the spinal cord. The relatively marked change of the nerve-cells and their processes, as compared with other elements, and often observed in general paralysis, is also emphasised by Dr. E. C. Carter.

To establish the atrophy of nervous elements as a *primary* affair, to which any vascular and neurogliar changes found are secondary or complicating, would be to revolutionise the antecedently predominant conception of the pathological processes of general paralysis. For, with (*a*) the general conception of a primary overgrowth of stromal connective tissue or lymph apparatus, whether of inflammatory or of other origin, aggressively producing *secondary* degeneration and atrophy, or destruction and disappearance, of the parenchymatous nervous elements—contrast (*b*) the contrary view of a *primary* degeneration, atrophy and disappearance of nervous tissue; other lesions present being secondary (or accidental); so that, to refer to the extreme pole of this opinion, Wernicke even held the huge spider cell overgrowth as being passive and compensatory, to fill the void left by the vanished nervous structures.

Some time before I first knew (1885) of Tuczek's results, through his work published in 1884, I held the view that, in some cases at least of general paralysis, the nerve-cell-and-fibre apparatus of the brain probably suffers earliest, and I maintained this view in a paper read at the annual meeting of the British Medical Association in 1883, and reproduced some of the conclusions drawn therein in my work on "General Paralysis,"[1] from which the following extracts are taken, the parts which more especially bear on the point at issue, being italicised now, but not in the original.

"It must be admitted that in many cases of general paralysis, all one sees may fairly be explained without calling in inflammation as a factor; and that, besides hyperæmia or its traces, and atrophy and degradation of true [nervous] elements, some substitution of a texture or substance of altered and lowered type, vital character, degree of development, and differentiation; for and in place of the usual texture of the part. . . . is in some cases practically what we see."

"Then if we enquire where the starting-point of the change is in general paralysis, our histological investigations would, on the whole, refer us[2] to the vessels and interstitial tissue, or at least intercellular elements. *Nevertheless, it may well be that the starting-point of the trouble is often in the ganglionic nerve-cells,* and that the excessive action, over-strain, and morbid stimulation or irritation by which the nerve-cells are brought into an invisible[2] state of damage, injury, lowered vitality, . . . superinduces. . . . active afflux of blood. . . . An equal degree of departure from a healthy condition existing in nerve-cell, in vessel, and in intercellular substance, the last two would give vastly more obvious histological indications of it than the first would. In the nerve-cell is an excessively delicate organisation. . . . Thus then, the effects of severe disturbance, and the resulting comparatively gross morbid change will first be *visible* in the vessels and interstitial (intercellular) substance."

---

[1] "General Paralysis," second edition, 1886, p. 340-342.

[2] *I.e.*, by the microscopical methods chiefly in vogue before then.

"On the whole, we may view general paralysis as essentially commencing with hyperæmia, and ending with chronic cortical degenerative cerebritis, and, usually, embryonic- and connective-tissue substitution, the change, *fundamentally parenchymatous*, affecting all the elements of the part, but usually under the methods of examination *hitherto chiefly in use*, presenting a more obvious and more marked affection of the blood-vessel walls and of the interstitial elements. . . . The cerebral, or even encephalic, are almost invariably associated with lesions of other parts of the cerebro-spinal nervous system; and the morbid action begins exceptionally in parts other than the cortex cerebri. Obviously this view allows of several varieties of general paralysis."

And it will not do to make too much of one factor in the morbid histology of general paralysis; and we have satisfaction in noting that Dagonet also recognises both inflammatory and sclerotic lesions as well as the degenerative and atrophic. Besides a vulnerability or selection of nervous elements in it, there is also a vulnerability or selection of blood-vessel walls; the circulatory troubles and lymphatic stasis tell for much, and one must restate that in cases of general paralysis, taken collectively, the lesions are multiple, complexly associated, and may earlier and more affect certain of the tissues composing the brain in some cases or parts, and others of them in others. May we then hope to divide general paralysis into varieties on this particular, and chiefly microscopical, anatomico-pathological basis? There are some indications that such may be the case, or that at least some light may thus be shed on the subject. In my book on "General Paralysis," besides what is said as to alcohol, syphilis, and lead, &c., is reference to a toxic origin as being probable in some cases; and in my article on general paralysis in the "Dictionary of Psychological Medicine," vol. i., p. 539, it is suggested whether the *primary* destructive change and disappearance of cortical nerve-fibres in general paralysis, and especially associated with a history of drink, "may be due to a form of alcoholic nerve-degeneration or neuritis, akin to alcoholic peripheral neuritis;" by which I mean that alcohol (and other toxic agents) probably may occasion primary nerve-fibre degeneration, or neuritis, not only in the peripheral nerves, but also in the nerve-fibres at the other peripheral pole of the nervous system, namely, at the periphery of the encephalic mass—the brain.

Some, especially recent, observations and conclusions seem to give support to the general view a moment ago suggested.

For example, long ago I published cases which, especially now, would be termed syphilitic general paralysis, or pseudo-general paralysis, in which the smaller blood-vessels (especially arterioles) of the cerebral cortex, were earlier and more markedly affected than the other elements.

The results of Bevan Lewis[1] may be of some value on this point, as assisting to indicate cases of general paralysis in which alcohol is one, or the chief factor; namely, that in chronic alcoholism, as compared with general paralysis, there is a somewhat different situation of the points of chief incidence of spider-cell development; and in the blood-vessel the morbid change is centred in the atheromatous state of the inner coat, the outer or adventitial investment not showing the enormous degree of nuclear proliferation found in general paralysis.

Raymond[2] also finds syphilitic general paralysis to be a chronic interstitial inflammation, having its point of departure in the capillaries of the brain, and more particularly of the cerebral cortex. Following the nomenclature of Gilbert and Lion, the infiltration by embryonic cells leads him to designate as diffuse embryonic meningo-encephalitis; or again, he names as diffuse vascular meningo-encephalitis; the brain-lesion of at least syphilitic general paralysis, if not of all forms. In the cortex, and less so elsewhere, the lymphatic sheath of the capillaries, and the lymphatic sheath and the wall of the larger vessels, show infiltrating accumulations of cells with rounded nuclei (nodular or diffuse periarteritis, ordinary secondary obliterative endarteritis ; in outer—musculo-connective—venous walls, similar diffuse or nodular infiltrations).

---

[1] "A Text Book of Mental Diseases," 1889, p. 538.

[2] *Archives de Neurologie*, Jan., 1894.

This, with considerable disappearance of tangential fibres, appears to be the earliest change; meningitis co-exists ; sometimes with miliary gummata, and often change of the large blood-vessels at the base of the brain, or branching therefrom.

R. Collela[1] concludes that, in the brain :—

(1) In general progressive paralysis with syphilitic infection, the histological changes chiefly affect the blood-vessels ; the neurogliar cells [increased size and number of spider cells] ; and the cellular protoplasm and protoplasmic processes of the nervous elements. The axis-cylinder processes [and nerve-fibres] are not destroyed, except in a few elements, and only later on. The changes begin essentially in the vascular network.

(2) In paralytic dementia with alcholic intoxication, we plainly observe a hypertrophy [but no notable increase in number] of spider cells ; and different phases of regressive nutritive disturbances in the nervous (axis-cylinder) processes. Blood-vessels healthy.

(3) In alcoholic insanity the alteration is essentially parenchymatous, affecting the nervous processes, and with a scarcely appreciable implication of the ganglionic bodies and protoplasmic processes of nerve-cells. Neuroglia and vessels healthy.

At all events, however this may be, further research on this line, promises to be more fruitful than a premature attempt to distinguish true general paralysis from the various forms of so-called "pseudo-general paralysis" by fine microscopical points alone. Thus, we find recent observers who speak of the pseudo-general paralyses as being analagous or else identical; or as being almost identical; *clinically*, with true general paralysis, and sometimes only distinguishable from it microscopically; proceeding to set up as a criterion that true general paralysis, as shown by its vascular lesions,* is, rather, an inflammatory affection, in the cerebral cortex at the least—a true encephalitis—and by this alone distinguishable from the pseudo-general paralyses; inasmuch as in these, on the contrary, the lesions are degenerative and regressive.

---

[1] From Golgi's Laboratory. *C. R. Acad. des Sci.*, Feb., 1893.

But when we turn to their account of the microscopical changes in true general paralysis, we find an account (and a correct one) of degenerative alterations in the cortical nerve-cells, proliferation of small round cells in the pericellular spaces, destructive changes in cortical and convolutional nerve-fibres. These changes give no active support to the criterion put forward. But it is the vascular lesions on which reliance is placed for this purpose, and these lesions are stated to be inflammatory and not degenerative; the new formation of vessels is maintained as a fact; and considerable diapedesis in the lymphatic sheath is said to cover or conceal the middle coat. On the other hand, degeneration of muscular coat and large yellow granulations are declared absent. So also is atheroma, if the true general paralysis is not "associated," that is to say, unless its lesions and those of pseudo-general paralysis co-exist.

Now, we shall see presently that vascular degeneration may exist even as an early change in true general paralysis, and thus the alleged criterion appears to fail.

At and from this point, so far as concerns this part of the subject, namely, the cerebral pathological anatomy, we take up exclusively the recent work of Professor Binswanger on the pathological histology of the disease of the cerebral cortex in general paralysis, especially in acute and early forms thereof.

Speaking generally, two series of chronic processes are observed in general paralysis:—

(1) Degenerative—atrophic changes in the specific nerve-tissue.

(2) Swelling, growth, and shrinking in the vascularized sustentacular apparatus.

As regards its lesions, the ground-substance takes a position intermediate to the two just mentioned, inasmuch as it shows both atrophic-degenerative and proliferative phenomena in the advanced forms of general paralysis.

Inflammatory processes in the narrower sense (exudative processes) develop after tissue lesions which call forth reparative processes of new formation of tissue from fixed cell-elements (not leucocytes).

Studying the acute and early forms of general paralysis, he discusses, in order, the several constituents of brain.

Nerve-fibres and nerve-cells.—The typical cases of general paralysis derive from chronic diffuse atrophic degenerative changes of the functionally active cortical substance (nervous elements). Probably these changes first affect the finest terminal ramifications of the medullated and non-medullated fibres (not by our present available means demonstrable with sufficient clearness); but even in the commencing stage of the disease these changes pass over to the medullated nerve-fibres (demonstrable by the staining of the medullary sheath), and to the ganglionic nerve-cells. How far structureless and structural elements of the original (ecto-dormal) ground- and supporting-substance take part in the disappearance of tissue cannot be shown with certainty: only at the nuclei of the glia-cells are the signs of degenerative processes recognisable.

Blood-vessels: distension with red blood corpuscles, hyaline degeneration, reparative thickening of tunics, growth of endothelial nuclei, narrowing of vessel-lumen.—Already in the early stages are the resultant phenomena of these tissue lesions clearly expressed in the blood-vessels. The venous channels are throughout much dilated, distended with red blood corpuscles, while there is not an essential increase of the leucocytes within these vessels. Already, also, does the vessel wall succumb to early regressive alterations, which particularly affect the arterioles and capillaries, and have the character of hyaline degeneration.

At the same time active reparative processes occur at the endothelial-adventitia of the vessels—beginning at the veins—processes which consist of a thickening of these tunics and increase of the endothelial nuclei. A part of these new formations of tissue also undergoes hyaline degeneration in the later stages of the disease. In other vessels, the proliferative processes attack the true vessel wall later on, and bring about its streaky thickenings and nuclear increase. Thereby the lumen of the vessels is narrowed, but complete obliteration of vessels—as observed in large numbers in old cases dying in the final period—is not demonstrable in the early stages. Neither in recent nor in long-standing cases could a new formation of vessels be established with certainty.

Lymph channel system (*of blood-vessels*).—In the early stages the system of intra- and extra-adventitial lymph-ways is already extremely dilated.

(*a*) Ampulla-like expansions of the *intra*-adventitial lymph space contain varying quantities of red and white blood corpuscles, coagulated lymph, amorphous, clumpy and finely granular blood pigment. Only few collections of leucocytes within the adventitial sheath are found in the early stages. Extravasation of constituents of the blood, particularly of red hæmacytes, as well as the relics of former such effusions, occur the most frequently in cases presenting wide-spread degeneration of the vascular walls.

In those early forms of general paralysis which take a slow course, uninterrupted by "paralytic attacks," until death happens from intercurrent disease; namely cases which constitute his first group; the accumulation of leucocytes in the endothelial sheath never reaches such a high degree and general spread as to justify the conclusion that exudative processes had existed during life. On the contrary, those leucocytal crowds are only to be taken as results of long-lasting and oft-repeated venous stases.

(*b*) Dilatation obtains throughout the *extra*-adventitial lymph-way network, including here the pericellular spaces. The contents of the extra-adventitial spaces are found to be much the same as, though less in amount than, those of the intra-adventitial already mentioned. An essential increase of lymphoid elements is not established for the lymph spaces of the cortex and the pericellular.

Stroma : scaffold- or interstitial-substance.—As far as is recognisable by the present methods of investigation, there are not processes of growth or increase of the fibrous parts of the interstitial framework. In the commencing stage it is only in the first or outermost layer of the cortex that (beside atrophic patches) there are evinced distinct increase and firmer quality of the glia-fibres. In the early forms of the disease the mesodermal supporting cells (spider cells) are not increased in the middle and deeper cortical layers. A distinct increase of these cells first appears in the most superficial cortical stratum.

Inflammation, paralytic attacks.—In the later course of general paralysis, and supervening on the changes already named, are true inflammatory, that is to say, exudative processes; which not seldom manifest themselves clinically by febrile action, states of somnolence, phenomena of cortico-motor irritation and paralysis ("paralytic attacks"). Should death follow in such an acute seizure, there are extensive small-cell accumulations in the intra- and extra-adventitial lymph way system. Yet must it be noted here that by no means all so-called paralytic attacks have this pathologico-anatomical basis; much rather may we hold that fluctuations in brain pressure, alone, represent the factors of paralytic attacks—fluctuations which are called forth by the hindered outflow of the lymphatic fluid overfilling the lymph channels of the brain. This will happen the more easily the more intensely the cortical functions are damaged by the diffuse disappearance of (nervous) tissue.

Pia mater; adhesions. Lymph stasis.—Participation of the pia in the morbid process plays an important part in the further development of general paralysis. In the early forms of that disease the pia is thickened and beset with numerous endothelial nuclei over the cerebral convexity, but only slightly so, and limited to isolated situations on the frontal lobe.

But the thickening and nuclear increase of the endothelial-adventitia, already mentioned, regularly appear in the vessels radiating into the cortex. . . . Both in the sphere of the vessels permeating the cortex and in the thickened pia, occur partial obliterations of the epicerebral space by partial cohesions of the pia with the cortex. These cohesions are produced, on the one side by ingrowth of the glia fibres into the pia; and on the other side by the spreading and growth of the endothelial septa, which, springing from the pia, traverse the epicerebral space. By the cohesion of the endothelial-adventitia with the neighbouring cortex-surface, those apertures of the extra-vascular lymph canal system which subserve outflow are closed, and thereby is produced an increased engorgement with fluid, particularly in the lymph spaces of the outermost cortical layer, but also found in the more deeply situated parts of the cortex. The transformation of perished tissue-materials to coagulated necrotic masses is wrought by the pathologically increased and altered lymph fluid.

Acute galloping general paralysis.—The so-called acute galloping forms of general paralysis are not distinguished from the typical form by any especial necroscopic condition. They are distinguished only by a more rapid progress of the pathological processes. The degenerative as well as the reparative processes gain a quicker and more diffused propagation. In some of the cases death occurs on the basis of the clinical phenomena conditionated by the tempestuous course of the disease; in which violent motor excitation, defective food-taking, exhaustion, broncho-pneumonia, intestinal catarrh, &c., play the chief part. True exudative inflammatory phenomena may then be entirely absent. The same conditions occur more markedly in another set of cases.

Whether it is the typical case of chronic course, or the galloping example of general paralysis, the diffuse pathological process first and chiefly affects the frontal lobe (including the insula) and parietal lobe, and then invades the temporo-sphenoidal and occipital regions. Early extensive participation of the occipital lobe has not been observed by him.

III.

SOME OTHER RECENT STUDIES IN GENERAL PARALYSIS.

A.—THE SPINAL LESIONS IN GENERAL PARALYSIS AND IN TABES; A COMPARATIVE STUDY.

Some have held that the spinal disease in general paralysis and tabes is one and the same; others have held them not to be identical. Joffroy maintained that both general paralysis and tabes are of parenchymatous origin, but that the former is not a "systematic" disease, whereas the latter is so, and of the sensory system. Like some others of us, Marie[1] finds that general paralysis rarely supervenes on tabes; a more frequent occurrence or sequence is the appearance of symptoms of lesion of the posterior columns, either simultaneously with, or in the course of, general paralysis.

Irrespectively of the question of precedence, also, the statistics of G. Renaud show only about 3 *per cent.* of general paralytics with clear symptoms of tabes; about 2 *per cent.* additional presenting more or less pronounced signs of sclerosis of the posterior columns, but insufficient for the diagnosis of tabes.

Marie claims that he had shown, in a previous communication, the differences in site between the lesions of the posterior columns in pellagra and in incipient tabes; in the former (pellagra) the posterior internal zone and the comma-shaped bundle chiefly suffering; whereas in the latter the parts mainly affected are the posterior nerve-roots, Lissauer's tract, the "cornu-radicular" zone and the nervous network of Clarke's columns. Therefore, he terms the former of "endogenous" origin; the latter of "exogenous"; the one change starting within, the other without, the cord.

Making, on the same lines, a comparative study of the lesions of the posterior columns in general paralysis and tabes, and using the figures published by Westphal as a representative test of the leading type of posterior-column change in general paralysis, he finds that the "cornu-radicular" zone is *not* altered (it is the external part of Burdach's column abutting on the middle third of the internal border of the posterior horn, and represents approximately the posterior root-zone). On the other hand, in the posterior columns, the comma-shaped fillet is affected: and so, also, is the "median zone of Flechsig," bordering on the posterior median fissure. The change is poliomyelitic, and is of "endogenous" origin.

---

[1] *Gazette des Hôpitaux*, January 16, 1894, p. 55.

These same parts of the posterior-external and posterior-median columns (altered as described in general paralysis with this first type of spinal change), are also affected with descending secondary degeneration in transverse lesions of the grey substance of the cord.

The source of these alterations of the posterior columns in general paralysis he seeks in a poliomyelitis, affecting the so-called "cells of the posterior columns," situated in the grey medullary substance, and described by several recent observers.

But, besides the affection of the posterior columns, there is, in general paralysis, usually, also, a sclerosis of the lateral columns of the cord. He thinks this lesion is not analogous to the secondary degeneration of hemiplegiacs, inasmuch as: (*a*) it is not wholly confined to the crossed pyramidal tract, but invades the neighbouring parts; as (*b*) in some cases the lesion of the crossed pyramidal tract ceases at the level of the medulla oblongata, and cannot be traced above; and as (*c*) the direct pyramidal tract is not similarly lesed.

Various views have been held as to why the crossed pyramidal tracts should degenerate in general paralysis; and Marie holds that the reason is to be sought in the centres on which they depend; that the pyramidal degeneration in general paralysis is analogous to that of the same territory in amyotrophic lateral sclerosis; and that this degeneration of the crossed pyramidal tracts (except for the greater or less part which a morbid cerebral process may or may not take therein) is chiefly produced by the alteration of certain cells, situate in the grey medullary substance of the cord, namely, the so-called "cells of the lateral column." On this view, the degeneration of lateral columns in general paralysis would be chiefly due to a poliomyelitis affecting certain special elements of the grey.

Thus, in general paralysis, two groups of lesions in the posterior and lateral columns, or combined degenerations or scleroses of both columns, are declared to be dependent upon systematised poliomyelitic changes.

Nevertheless, this combined type is not the only one in general paralysis. The lateral columns alone may be affected, or the posterior columns only.

If the posterior columns only are affected, the lesions are much more like those of typical tabes in situation; *e.g.*, the "cornu-radicular" zone is affected, and the pathological process is "exogenous."

These cases are different from those of the usual type above-described in general paralysis, not only anatomico-pathologically, but also clinically; at least, those are so in which a lesion of the posterior column has for a long while preceded the cerebral symptoms. And, as regards cases such as these last, Fürstner, like others of us, has observed, and has emphasised, the protracted course of the disease; the prolonged retention of mental integrity, and, later on, the unusual degree of preservation of considerable mental power; the comparatively slight affections of speech, tongue and face; the relative rarity of "paralytic attacks"; and he questioned whether these are really examples of general paralysis. Especially in some instances is it difficult to classify certain of the cases of this group, namely, those with the symptoms of tabes still rudimentary only, even after several years' duration, there being merely reflex iridoplegia, absent knee-jerk, hypalgesia, slight bladder trouble; and, on the mental side, only a moderate diminution of memory, some isolated indications of mental deterioration, and a state of apathy. Therefore, overt tabes does not readily turn into general paralysis.

To sum up, then, there are two main great distinct types; one commencing within the cord and poliomyelitic in origin; the other commencing without the cord, and invading by way of the posterior nerve-roots.

Finally, these two types, limned above, may be combined in the same patient, some of the lesions of the posterior columns in such a case being "exogenous," and others "endogenous,"[1] and associated with the lateral column lesion of the first type.

On this view, the spinal and other lesions of general paralysis would be parenchymatous in origin; the toxic agent producing them acting primarily on nerve-cells; namely, on the nerve-cells of the brain, on the nerve-cells of the grey matter of the cord for the so-called "endogenous" changes of the cord, and on the nerve-cells of the spinal ganglia for its "exogenous" changes.

In reference to the foregoing, I think Dr. Marie presents the matter in too schematic a way, limits his view far too much to certain types of lesion, and does not adequately acknowledge the effect of the cerebral lesions of general paralysis in occasioning secondary spinal degeneration, nor sufficiently allow for the great variety, and often diffuse character, or irregular distribution, of spinal changes in general paralysis. (See the present writer's work on the subject; *op. cit.*, pp. 288 and 322.)

---

[1] Using these words in the sense already described.

Moreover, the two cases reported by Westphal, and taken by Marie as crucial examples of the lesions of the posterior columns in two different groups of cases forming types of spinal lesions in general paralysis, and occupying different situations in those columns; and the one of "endogenous," the other of "exogenous" origin; differed comparatively little as concerns spinal symptoms; for none of them were ataxic, and the loss of knee-jerk was the one spinal feature in common, and from it alone was posterior columnar change diagnosed.

Nor does either of Westphal's cases, chosen as typical, correspond closely in posterior columnar change with the other case in the same paper, or with the case depicted in a subsequent paper by the same observer in the same publication. This last, indeed, resembles more the tabetic type of distribution of chief spinal change.

The presence or absence of ataxy in cases of posterior spinal lesions in general paralysis, Westphal himself was inclined to make dependent on the intensity of the disease (the degree of atrophy), and on the greater or less participation of the nerve-roots therein. And he laid some stress on the fact that those lesions as found in general paralysis are often unaccompanied by marked, or by any, motor or sensory symptoms.

B.—SPINAL FORMS OF GENERAL PARALYSIS.

It has been widely recognised that some cases of general paralysis begin, or appear to begin, with spinal symptoms, the onset seeming to be by the spinal cord or spinal nerves. Such cases are often called examples of the spinal form, or forms, of general paralysis. General paralysis tends to affect the whole nervous system, but its earlier incidence may be on this or that part of it; and may occasion clinical appearances which call to mind those of various diseases of the spinal cord.

A study of the spinal lesions and symptoms in, and spinal forms of, general paralysis, has just been published by Dr. M. Klippel.[1] As space is limited and time presses, the spinal clinical forms will be briefly noticed, and then the lesions.

The spinal clinical forms of general paralysis (in the above sense) are multiple: some take a tabic aspect, sometimes the early symptoms are those of bulbar paralysis, of lateral sclerosis, of muscular myelopathic atrophy, &c. These symptoms are determined by localisation of the lesion more particularly on this or that system or part of the cord, or bulb; and, for purposes of description, the forms must necessarily be more individualised, and less mixed or "combined," than they actually are in fact. Owing to this mixture or association of lesions, the special symptoms do not usually correspond directly in degree to the degree of lesion producing each; the clinical manifestation of the lesion in this part being perhaps modified or neutralised more or less by the lesion in that.

---

[1] *Archives de Médecin Expérimentale*, t. vi., Jan., 1894.

Cases of spinal disease, which, established as such, afterwards become *complicated* with general paralysis, are not included here.

Klippel prefers to divide the spinal, into five forms:—

(1) *The tabetic form.*—With the sclerosis of the posterior columns there is usually also that of the lateral columns. He says the symptoms are, in a word, those of tabes. But, in fact, the symptoms are often only those of an incomplete, or partial, or rudimentary tabes, or tabetiform state.

Besides this form, and to be distinguished from it, are the cases of tabes with cerebral lesions (Jendrassik), but only analagous to, not identical with, those of general paralysis (Klippel). Also, established tabes, late and slowly becoming complicated with true general paralysis, must be kept separate from the form we are considering.

(2) *The lateral sclerosis form.*—Whereas in the first form the lesions chiefly affect sensory elements or conductors of the cord; in this second form the chief incidence is on the motor paths.

Bi-lateral pareses, more on one side; some appearances of muscular contracture, and of exaggeration of reflexes, are the main symptoms; but often are transitory only; the invasion reaching other parts of the cord. Posterior sclerosis is usually conjoined with the lateral.

(3) *The amyotrophic form.*—The state precedent to, and forming the onset of, general paralysis in this form takes either: (*a*) that of progressive muscular atrophy of the Aran-Duchenne type, or (*b*) that of amyotrophic lateral sclerosis (lesion of lateral columns [part of], also of anterior horns, and peripheral nerves).

(4) *Bulbar forms.*—In one case he observed symptoms of bulbar paralysis in a patient whose mind did not seem to be affected, but who three months afterwards showed dementia and the usual indications of general paralysis.

(5) *Neuritic forms.*—As, for instance, neuritis of the limbs with muscular atrophy; especially forming the mode of onset of some cases of traumatic general paralysis.

These are convenient divisions, but do not seem to be absolutely necessary, if we bear in mind the principle involved in them, which has been much debated, but accepted by many.

For the most part, the spinal lesions in general paralysis are situated in some part, or parts, of the posterior half of a transverse section of the cord. They are, in a certain degree, diffuse; and yet present some character of systematisation, affecting, as they do, the sensory or motor elements or both, of a physiological system.

In the posterior columns, there may be only simple thickening of the connective framework, leading by compression to simple atrophy without degeneration of the nerve-fibres; in other cases amidst the sclerous, or seemingly œdematous, tissue are nerve-tubes with granular degeneration of the myeline sheath; in other cases, or in parts of the lesion, the destruction of the nervous elements is extreme. Differences in the site of the lesions present themselves in different cases. The blood-vessels of the affected parts are dilated and are gorged with blood.

Much the same description applies to the lateral columns; and the sclerosis here is not directly as that of the posterior columns (when they co-exist). And often the direct pyramidal tract is exempt, as, to a large degree, also, the rest of the anterior columns usually is.

In the grey matter of the cord are often important lesions of the ganglion nerve-cells and of the nerve-network. Those of the cells may be simple atrophy or degeneration, and in some cases even disappearance of the cells. The cells of Clarke's column may be similarly affected. There may be destruction of the nervous network of the grey substance and Clarke's column.

In the nerve roots, the lesions chiefly affect the lumbar and sacral ones, and are associated with signs of meningitis; in the cervical and lumbar roots is an abnormal number, relatively, of fine slender fibres.

Simple atrophy, chiefly, affects the nerve-roots; degenerative neuritis the peripheral nerves.

The course of the lesions consists in a degeneration and absorption of the myeline-sheaths; a consecutive congestion and exudation in some cases, "the exudative or myxomatous form of the lesion," as he calls it; and in other cases, and as a third stage, this, *plus* a secondary inflammatory process—"the inflammatory form." Thus the change in nervous elements taken here to be primary, is elsewhere in the article described as secondary.

Pathogenetically, in the cord is a double morbid process :—

(1) Foci of myelitis, developing there in many and different points.

(2) Dystrophy of nervous elements (especially lateral columns), dependent upon cerebral lesions and influences.

On the spinal *symptoms* there is nothing new or that needs mention.

To some extent, the same, or analogous, remarks just made on the paper of Dr. Marie apply here also.

Cases have recently been reported from the clinic of Prof. Joffroy, of Paris, exemplifying "the spinal forms of general paralysis," *i.e.*, in which spinal symptoms or diseases precede overt, recognised cerebral symptoms of general paralysis of the insane. (*Rev. de Med.*, 1894).

In one case the chief symptoms, as of a true tabes dorsalis, had been manifest two years previously to the oncoming of cerebral symptoms of general paralysis, and the latter was associated with a very manifest improvement in the gait. At the necropsy, were found the cerebral lesions of general paralysis. The spinal cord was scarcely changed, macroscopically, but, on microscopical examination revealed (not the localisation as in true tabes, but) a diffuse myelitis of posterior and lateral columns. Therefore, the case was not one of tabes associated with general paralysis, but one of general paralysis commencing at the spinal cord and with tabetiform symptoms.

Two other cases without necropsy are taken as having the same significance.

M. Joffroy states that the association of true tabes and general paralysis is more rare than "extremely rare." As to this, I may say that about one per cent. of the cases of general paralysis are preceded by tabes dorsalis.

He also briefly mentions a case of progressive muscular atrophy preceding general paralysis by several years (the so-called amyotrophic spinal form of general paralysis). Like others, he very properly separates this from the *late* muscular wasting, so frequent in general paralysis.

Another case is mentioned, in which occurred an arthropathy of the knee, which he terms rheumatic, but which was, apparently, tabic or taboid in nature. Three years afterwards, were symptoms of the onset of general paralysis.

Besides these cases of so-called spinal forms of general paralysis simulating tabes, progressive muscular atrophy, and "rheumatic phenomena," are mentioned those in which the simulation is of spastic spinal paralysis, and possibly, of cerebrospinal insular sclerosis.

M. Joffroy has, therefore, seen, or known of, an interesting set of cases belonging to what some call the spinal forms of general paralysis; really meaning thereby the part affected by its incidence at first. These cases are welcome illustrations of facts already established as regards general paralysis; and, although there is nothing new in them, they offer three points as to which they present accentuation of certain features observed in other previously reported cases:—

(1) That (excepting the amyotrophic) he appears to have observed a somewhat unusual frequency and degree of improvement in the spinal symptoms, of such cases, when the cerebral of general paralysis appear. But the observation of such improvement is an old one; examples of the kind having been published more than thirty years ago.

(2) That in the case with necropsy, the simulation of antecedent tabes was more marked than it usually is in instances of the same kind.

(3) That the knee-arthopathy of a case mentioned was more than usually early in appearance (if really connected with the general paralysis at all, which, owing to the brevity of the report, is a quite uncertain point).

C.—THE OCULAR DISORDERS IN GENERAL PARALYSIS.[1]

Of these, the chief disorder is gradual and progressive internal ophthalmoplegia; paralysis of the constrictor of the pupil and of the ciliary muscle, the former contracting to light and in efforts of accommodation; the latter acting only in accommodation and adapting the eye to distances. Hence a triple research is necessary, by the usual methods; namely, to ascertain the condition of the reaction of iris to light; secondly, the reaction of the iris and pupillary change in accommodation; and, thirdly, the power of ciliary muscle in visual accommodation. As M. Gilbert-Ballet mentions, his general results confirm the conclusions on this subject long ago published by the present writer, which were as follows[2]:—

"Speaking in general terms, I think the course of events is usually this. The pupils become somewhat sluggish to light, this increases and some failure of their movement in accommodation often supervenes, the tendency being for the one failure to follow the other at a considerable interval. The latter failure, however, may not follow; and with immobility to light, but normal activity in accommodation, reflex iridoplegia, pure and simple, is present; or, as one might say, iridal reflex monoplegia, failure of the contraction of the sphincter iridis under the influence of light or stimulation of optic nerve. If and when, to this reflex iridoplegia there is added cycloplegia, or failure of ciliary muscle in accommodation, internal ophthalmoplegia is present. With [this] cycloplegia usually comes failure of the contraction of sphincter iridis during accommodation"; which, indeed, is an integral part of a complete internal ophthalmoplegia.

---

[1] M. Gilbert-Ballet. "Les Troubles oculaires dans la Paralysie générale progressive." *Le Progrès Médical*, June 10, 1893.

[2] "General Paralysis of the Insane," second edition, p. 109.

M. Gilbert-Ballet has worked out this ocular failure in greater detail, on the same lines. For one eye, considered separately, he has arranged four degrees of it: (1) paresis of movements of reaction to light; (2) abolition of these movements; (3) abolition of them, and paresis of accommodation; (4) complete paralysis both to light and in accommodation.

For the two eyes, considered conjointly, five degrees are similarly arranged: (1) loss of reaction to light only; accommodation normal; (2) both of above forms of iridal reaction weakened; (3) no reaction to light and accommodation in the eye with the more dilated pupil; simple reflex iridoplegia in the other eye; (4) same as "(3)" with weak reaction of second eye to light and accommodation; (5) complete bilateral internal ophthalmoplegia.

These ocular failures sometimes considerably precede the other manifestations of general paralysis. The nerve fibres, to iris and ciliary muscle concerned in these symptoms, come immediately from the common motor oculi nerve, which also supplies the external ocular muscles, which usually are unaffected in general paralysis. By a process of exclusion, it is concluded that the lesion, on which these symptoms depend, affects the particular nuclei of origin of third cranial nerves from which the nerve-fibres in question arise; and that it is the nuclei composing the upper group of nuclei (of third nerves), namely those whose site is the wall of the third ventricle, which—either directly or through contiguity—are mainly thus affected by the morbid process of general paralysis; whereas the other nuclei in the inferior part of the Sylvian aqueduct ordinarily escape. And when paralysis of external ocular muscles does occur in general paralysis, it is usually in those cases which are preceded by tabes; and frequently it is transitory. Nystagmus is occasionally observed;

and so is atrophy of optic disc, but, like Dr. Gowers, M. Ballet makes light of the ophthalmoscopic changes in general paralysis, which, as a rule, he finds to be slight or absent.

D.—Juvenile General Paralysis.[1]

Dr. Toulouse has collected sixteen cases of juvenile general paralysis, published by others. Fifteen years was the average age of the patients at the onset of the disease. Males were affected twice as often as females;[2] and the majority belonged to families of the labouring class.

With regard to morbid hereditary antecedents; of the fourteen cases where these were known—and counting each of them as often as it was found either with different, separate patients, or with the same patient—there was an ancestral history in six instances, of alcoholism; in four, of syphilis; in three, of mental disorder; in two, of general paralysis; and in one, of "congestive" disease.

Acquired syphilis and cranial injury have been indicated by other observers. And there were some indications of congenital syphilis in a case observed by myself.

From one aspect, juvenile general paralysis may be viewed as a precocious dementia; as the premature senility and death of a brain still young. Considered as a retrogression of the nervous elements of the brain, it is a defect of biological evolution attributable to the operation of the factors already mentioned, and perhaps to the disturbances and deep modifications stamped on the organism at, and by, puberty.

In juvenile general paralysis, at least some physical symptoms are apt to be relatively more marked than usual, more intense, and abrupt in onset; so that the youthful patients soon, perhaps, evince grave disturbances of gait, severe headache, convulsive and comatose attacks followed by paresis; also paralyses or contractures, grave emaciation, arrest of sexual development.

On the mental side, are predominantly intellectual enfeeblement and dementia; the dramatic deliria, so often observed in adult general paralysis, rarely occurring in pubescent or adolescent cases, or being slight if present.

Arrest and remission are less frequent than in the adult forms, but the course of the disease is in some instances slow.

---

[1] Dr. É. Toulouse, *Gazette des Hôpitaux*, August 24, 1898.

[2] This conclusion is drawn from twelve cases; it is not stated whence these were selected; and it is the reverse of that drawn by other observers.—W. J. M.

The lesions found are much the same as in the adult; atrophy being, possibly, a more striking feature.

The differential diagnosis must be carefully made from other somewhat like conditions occurring in youth. Anti-syphilitic treatment fails; in preventive management lies the only hope.[1]

E.—The Temperature in General Paralysis.[2]

The writers of the article next to be considered, conclude that in general paralysis the average bodily temperature, and the diurnal oscillations of temperature, correspond to physiological normes; that the same, practically, is true of the asymmetrical axillary differences; and that when unusual variations of temperature occur in general paralysis, their cause must be sought for in conditions not related to the pathological phenomena of that disease.

In reference to their conclusions, and to their criticism of preceding observers, one may notice that some of their cases are unsuitable, or are defectively suitable, for the purpose required. Moreover, they have made no observations on the variations of temperature taking place in connection with the paralytic and convulsive seizures in general paralysis; and yet these are the very conditions in which some of the most important and interesting variations of temperature occur in general paralysis. Besides the variations in connection with these seizures, I also often found alterations of temperature in connection with attacks of mental excitement in general paralysis; important changes and inversions of the temperature-curve in the twenty-four hours; in some conditions, subnormal temperatures; and that the temperature in general paralysis not seldom shows a preternatural mobility, a mercurial facility of change as the result of comparatively slight causes.

F.—Tumour-like Congenital Spinal Deformities in General Paralysis.[3]

A case of general paralysis is described by Dr. Bernhard Feist, in which, besides spinal cord changes resulting from the morbid process of that disease, there were several tumour-deformities of the spinal cord; partly at least, apparently of congenital origin. A number of plates accompany the record of the case, and clearly show the resulting deformity, displacement, or disjunction and separation, of several parts of the cord.

---

[1] Since the above was written, a contribution to this subject has been made in the *Journal of Mental Science*, January, 1894, by Dr. Middlemass.

[2] Dr. Fr. Peterson and Dr. C. H. Longdon, *Journal of Nervous and Mental Disease*, November, 1893.

[3] *Archiv. f. Pathol. Anat.* (Virchow's), Bd. 130, Hft. 3, p. 444.

G.—THE VISCERAL COMPLICATIONS OF GENERAL PARALYSIS.

Just as this article is going to the press, I see one by M. G. Durante on the above subject. He asserts that the visceral complications of general paralysis have been but little studied, and almost passed over in silence, even in the recent monographs on general paralysis. Had he done me the honour of consulting my monograph thereon he would have read at pages 294 to 299, second edition, 1886, a very minute record, expressed in percentages, of the various visceral lesions found in general paralysis in necropsies made by myself, and much larger in number than those he cites from M. Klippel's contribution dated six years later, *i.e.*, in 1892, and twelve years later than the fairly full and exact record of those lesions in the first edition of my work, 1880.

M. Durante's contribution is chiefly an exposition of the observations and conclusions of Dr. Klippel, who has carried the study of the visceral complications in general paralysis a stage further, and who has endeavoured to establish certain points, the two chief ones of which I will briefly mention.

One is, that among the tubercular lesions found in general paralytics there is a variety which has ætiological importance; that, in fact, some cases of general paralysis are due to the late or tardy, post-infectious sclerosis—occurring remotely and without tubercular nodules *in loco*—and induced by the chronic intoxication (of microbic origin), accompanying the tuberculosis, which last, contrary to the usual rule, has in these cases reached a fourth stage. This chronic intoxication probably exercising an elective affinity on certain tissues (here cerebro-spinal), determined by a hereditary or acquired predisposition.

In such cases the pulmonary tubercular disease is slow, chronic, often latent, fibrous. At the necropsy are old fibrous or calcified tubercles, or tubercles most of them fibrous; and sometimes old cavities with hard, dry, thick fibrous walls—old lesions which for a long while had been latent. As recent changes in the lungs, may also be tubercular cavities, disseminated tubercular granulations, or caseous pneumonia with rapid softening.

My statistics (*loc. cit.*) show tubercles, caseous masses, or cavities, in ·26 *per cent.* of general paralytics; traces of former cured tubercle in 12 *per cent.* If I recollect aright, without re-examining the records, the preceding would include some of the cases with lung induration or cirrhosis, found in 14 *per cent.* (4 *per cent.*, out of this 14 *per cent.*, presenting very marked cicatrices). Consequently it is at least clear that a considerable number of general paralytics have had chronic, slow, or else regressive, pulmonary tuberculosis, before they became general paralytic.

The other chief point is that the set of visceral changes *directly* dependent upon the lesions of general paralysis in the nervous system, especially in the cortex cerebri, is "vaso-paralytic"; and amounts to a chronic congestion due to chronic paralytic dilatation of the capillaries—a constant condition but undergoing attacks of transitory increase or exacerbation, in congestive strokes. This capillary dilatation and congestion tend to form punctiform hæmorrhagic foci by vascular rupture or diapedesis. Blood pigment deposits show the chronicity of the condition.

To this process these writers attribute the visceral alterations, specially and directly dependent upon the lesions of the (cerebro-spinal) nervous centres in general paralysis; as, *e.g.*, in the lung, chronic congestion in different degrees, including hæmorrhage, broncho-pneumonia in foci, and even the apyretic pseudo-lobar varieties thereof. Thus they employ the term congestion with an unusually wide and inclusive meaning, and two-thirds of Klippel's necropsies show congestion used in this over-wide sense. Sir J. C. Brown's statistics gave congestion in 49 per cent.

My published statistics (*loc. cit.*) showed 64 per cent. with very marked pulmonary congestion; *i.é.*, in which it was the dominant condition, or else existed highly-marked together with other lesions. But besides these, it was distinctly present in many other cases, associated with more important changes of which it was, at the moment, taken to be merely an incidental accompaniment or appanage; so that, altogether, there would probably be congestion of some sort and degree in 80 or 90 per cent., or more. Nevertheless, the above writers appear to me to ascribe a somewhat too great frequency and importance to pulmonary congestion as a *direct* symptom of general paralysis; and closer examination will show that in many cases the congestion has not that meaning, and is only an indirect result or symptom, and even sometimes an incident of little importance.

In relation to the subjects of this last section, see my work, *op. cit.*, Chapters viii. and ix., which deal with the vaso-motor and trophic disorders and defects in general paralysis; also pp. 335, 194, 190 *et seq.*, 294 *et seq.*, 270-5, 341, 352-3.—W. J. M.

H.—ARTHROPATHY IN GENERAL PARALYSIS.[1]

In correcting "proof" of the preceding article, I take the opportunity of adding a few lines on papers which have just come to hand.

A case of arthropathy in general paralysis, with necropsy, is recorded by Dr. Lloyd. The patient, a male, aged 38, had suffered for some years with pains in the legs, which had been called rheumatic; otherwise he was apparently healthy up to one year and a-half before admission, when he had a swelling of his knees. At this time no mental symptoms were observed. Six months before admission he began to have mental symptoms. He had delusions of persecution; he became silly and weak; had a remission; then a relapse. Drawling speech began three months before his admission. After admission were a maniacal episode, and later on a grandiose delusional state. The physical symptoms partook partly of general paralytic type, partly of tabetiform type, and partly of that of insular sclerosis. The knee-joints were enormously swollen; and apparently were in the ordinary state of tabetic arthropathy (Charcot's), and presented changes of both the atrophic and hypertrophic kind. Cystitis and pyelitis preceded death.

The adhesions and chronic meningitis chiefly affected the base of the brain and the frontal lobes.

"The cord presents the type of degeneration seen usually in forms of cerebro-spinal sclerosis . . . . it does not present a typical posterior sclerosis. It rather combines two system lesions: (1) partial posterior sclerosis; and (2) sclerosis of the motor tracts from one hemisphere [the left.]

"[Dorsal cord.] (1) The posterior sclerosis is not distributed symmetrically as in locomotor ataxia, to the lesion of which disease, indeed, it presents but a partial resemblance. A large sclerosed patch occupies the anterior end of the postero-median column and the contiguous part of the postero-external column of the left side, and, less extensively, the anterior part of the median column of the right side. The postero-external column of the right side escapes almost completely . . . . The posterior root-zones practically escape (?

"(2) The motor tracts from the left hemisphere alone are involved, and these not typically. The sclerosed area in the right lateral column is not exactly co-extensive with the crossed pyramidal tract, but is more extensive. At its periphery a line of less degenerated fibres marks the position of the direct cerebellar tract. The region of the direct pyramidal tract on the left side presents a fan-shaped area of degeneration, sharply demarcated, and extending from the horn to the anterior median fissure. It is not co-extensive with the direct pyramidal tract." No very marked changes in multipolar cells of anterior horns in the part of dorsal cord examined; those in lumbar region not examined. Some have attributed those arthropathies to degeneration of these cells.

---

[1] Dr. James H. Lloyd, *Philadelphia Hospital Reports*, 1892.

The above case was, at least, more or less unusual as regards the ordinary physical objective signs of general paralysis. There is not space to set this fully forth by quoting all about the physical aspects, therefore I have omitted them. But in reading it the impression comes that it was, possibly, one of insular sclerosis, or one of general paralysis *secondary to* insular sclerosis, whether or not the former arises strictly as an example of general paralysis by invasion from the vicinity; in other words, by extension by contiguity. It reminds me somewhat of some such cases published by Schultze. The above impression is not altogether set aside by the necropsy.

This subject I have dealt with elsewhere. In the above case the microscopical examination was limited to one part of the cord; and it is possible that, applied to other parts of the cerebro-spinal system, it might have revealed some scattered foci of sclerosis. And in the part of the cord so examined the change, practically, was as much, or almost so, of an insular sclerosis type as of a systemic type. Yet I fully admit the more or less "irregularly distributed disease-process," in general paralysis; indeed, I have used stronger terms in describing it, *e.g.*, "secondary scattered sclerosis." But if we accept the case as one of general paralysis, independent of insular sclerosis, there still remain the tabetiform (or tabetic) symptoms and lesions to be reckoned with. In this respect the case, however, harmonises pretty fairly with most of the few already recorded of arthropathy of the same kind in general paralysis, including two by myself (besides one not yet published).

I.—NEURASTHENIC FORM OF THE PRODROMIC STAGE IN GENERAL PARALYSIS.

M. Gilbert-Ballet[1] records several cases of general paralysis in which neurasthenic symptoms were prominent in the prodromic stage, and sometimes for many months, or even for some years;

---

[1] La période prodromique à forme neurasthénique dans la paralysie générale." *La Semaine Médicale*, 1894.

although in some cases they and the prodromic stage may be only of brief duration. The chief symptoms are headache, vertigo, obscurations, acute pain, dyspeptic disorders, hypochondriasis, general feeling of lassitude and of weakness. These, and the various other symptoms in the above cases, have all been very fully described before, as occurring sometimes in the prodromic stage of general paralysis.

The characters relied upon to distinguish these symptoms in the prodromic stage of general paralysis from simple neurasthenia are:—

In the former:—

(1) The absence of the stigmata of neurasthenia (helmet-sensation, back-ache, sacral patch, &c).

(2) The neuralgic pains; multiple, essentially mobile, and varying day by day (lightning pains, from spinal lesions, are excepted here).

(3) The sudden changes in the patient's state, *e.g.*, from a sad and downcast, despairing condition, to one of exuberant joy, satisfaction, forgetfulness of the past evils and sufferings, or assertion of recovery therefrom.

J.—The Morbid Anatomy and Pathology of the Neuro-Muscular Changes in General Paralysis.

In an excellent article, Dr. Alfred W. Campbell[1] communicates the results of his work on this subject. In all the cases examined he found the vagi nerves extensively and strikingly diseased, and this disease is connected with important clinical conditions. The phrenic nerves suffered also, but less.

In the mixed spinal nerves, and their peripheral terminations, the alteration appeared to be a combination of a parenchymatous degeneration, and an interstitial or adventitial inflammation.

In the anterior spinal nerve roots, and in the posterior roots, between the cord and the ganglia, was always a considerable degree of degeneration, but never extensive. "In the posterior root ganglia, no obvious degeneration of nerve-cells, beyond some hyper-pigmentation was noticed, and the posterior roots beyond the ganglia were but little diseased."

In the *muscles* were fatty degeneration, atrophy, and complete or partial disappearance of a number of muscle fibres, with proliferation and increase of the nuclei of the sarcolemma and connective tissue. The number of motor end-plates was lessened, some were in process of degeneration; in some muscles was Eichhorst's "neuritis fascians."

---

[1] *Journal of Mental Science*, April, 1894.

The above-described changes he brings, very naturally, into line with the neuro-muscular changes in multiple neuritis of the primary intrinsic toxæmic group: referring to cases of general paralysis taken to be "purely idiopathic," and not due to syphilis or alcohol, with their effects upon the peripheral nerves.

These observations go to support me in what is already in print in the earlier part of this communication, and elsewhere on a toxic origin of general paralysis.

W. JULIUS MICKLE.

Clinical Case.

A CASE OF PSEUDO-BULBAR PARALYSIS, DUE TO LESIONS IN EACH INTERNAL CAPSULE; DEGENERATION OF DIRECT AND CROSSED PYRAMIDAL TRACTS.

BY W. S. COLMAN, M.D., M.R.C.P.

Assistant Physician to the Hospital for Sick Children, Registrar and Pathologist to the National Hospital for the Paralysed and Epileptic.

THE following case was admitted into the National Hospital, Queen Square, on December 31, 1892, under the care of Dr. Hughlings Jackson, who has kindly allowed me to publish it. For the notes of the state on admission I am indebted to Mr. Stoddart, the clinical clerk.

William S., aged 49, a porter, says he was perfectly well until a year ago, when his wife noticed that he dragged his left leg in walking. This ultimately prevented his working, but at the end of two months he had recovered sufficiently to be able to resume his occupation; but before long the weakness of the left leg returned, and he was quite helpless.

Six months ago he was noticed to be speaking hoarsely, and the difficulty of articulation has increased up to the time of admission. He never had any difficulty in finding the right word with which to express himself.

Two months ago he had difficulty in swallowing, noticed especially with liquids. The right leg at the same time became stiff and powerless like the left.

From this time an alteration in his mental condition was observed. He would laugh or cry incontrollably at the slightest thing. He has had no hallucinations.

Previous history.—He denies syphilis and its sequelæ. His wife has never been pregnant. No history of alcoholic excess. He has been subject to much physical strain in his occupation as porter.

Present State.—Patient is a well-porportioned, muscular man, complaining of weakness and stiffness of both lower limbs, inability to walk, and difficulty of articulation.

Mental Condition.—He has a vacant expression, and is provoked by the merest trifles into loud laughter, ending in an inspiratory whoop, or in unreasonable weeping. He complains of loss of memory. His mental condition generally seems dull and confused, but his difficulties of articulation are so great that investigation is extremely difficult. He does not complain of headache or vertigo. He sleeps well at night, and there is no drowsiness during the day. No delusions or hallucinations can be elicited.

Speech.—He finds great difficulty in articulating words, to such an extent that he resents being made to speak, and prefers to express himself by signs. Phonation hardly ever gets beyond a hoarse whisper in speaking. He cannot pronounce the explosive consonants, and his speech is very thick, as if his tongue was too large and almost filled up the buccal cavity. There is no true aphasia.

Motor Symptoms.—There is weakness of the muscles of face, arm and leg on both sides, more marked throughout on the right side.

Face, &c.—There is distinct weakness of both sides of the ordinary cerebral type, and there is also considerable weakening of the occipito-frontales, and orbicularis palpebrarum.

The left side acts better than the right, so that the mouth is drawn to the left in laughing, and when he attempts to shew his teeth, the left naso-labial fold is more marked than the right. He is quite unable to whistle.

Tongue.—All movements can be performed, though defectively, *e.g.*, he can only prostrate the tip of his tongue as far as the lips. There is no obvious wasting of the tongue and no fibrilliary twitching.

Palate.—Soft palate, deficient both in voluntary and reflex contraction on both sides. (It afterwards became completely paralysed.)

Larynx.—Both vocal cords congested, but move well (Dr. Semon).

Neck.—There is considerable weakness of sterno-mastoids and trapezii and probably deeper muscles, so that he cannot sit up in bed without having his head supported.

Upper Limbs.—Almost all movements of both upper limbs can be carried out, although feebly. Movements are distinctly weaker on the right side.

Trunk.—Muscles of the back are very weak. He cannot sit up, even with the aid of his arms.

Lower Limbs.—Extremely weak, and occasionally rigid, but not always so. He is quite unable to stand.

Superficial Reflexes.—Plantars increased: abdominal and epigastric not obtained.

Deep Reflexes.—Knee jerks much exaggerated. Ankle clonus present on both sides. Wrist and elbow jerks increased. Jaw jerk present.

Organic Reflexes.—Automatic emptying of bladder. For some weeks before his death he also passed his motions unconsciously.

Sensation.—Sensation to all forms of stimuli was quite normal in every part.

There were no indications of disease in any of the thoracic or abdominal viscera. Four months after admission he was attacked with general eczema. He seemed ill, and his temperature rose to 101°. In a few days there were signs of general bronchitis, and he died from this complication on May 5, 1893.

Examination of the Brain.—After hardening the brain, &c., in Müller's fluid, the following conditions were found:—

The convolutions showed a little general wasting, but were otherwise normal.

The vessels at the base of the brain were atheromatous, but elsewhere they were natural and the membranes thin.

On making vertical sections through the internal capsules, they were found to be soft and friable. Under the microscope sections showed many bundles of completely degenerated fibres separated by normal bundles, at the point where the fibres streamed out of the capsule into the crus, and anterior to this were several small areas of softening. Many of the small arteries in this neighbourhood were completely thrombosed. In the right capsule was a patch containing hæmatoidin crystals, evidently the site of a small hæmorrhage.

The degenerations could be traced in the pyramidal tract to the crus, pons and medulla, and in the whole length of cord in the lateral pyramidal tracts, and as far as the third dorsal in the direct pyramidal tract. The degenerations were more extensive on the right side of the cord than the left.

The cells of the hypo-glossal nucleus and the anterior cornual cells of the spinal cord were not diminished in number, and were quite normal in appearance.

The other organs shewed no change of importance. In a case recorded by Drs. Hughlings Jackson and Taylor, in which I examined the cord, there were very similar symptoms and lesions in the same position, but in that case there was no degeneration of either direct pyramidal tract. I have been able to find no reason for the difference in the two cases.

Remarks.—The course of the disease in this case closely resembled one of bulbar paralysis, with descending changes in the cord. A close investigation of the history, in the light of the *post-mortem* examination, shows that there were two attacks of left hemiplegia, followed at a long interval by one on the right side, and coincident with this the development of marked bulbar symptoms, as in the cases recorded by Barlow,[1] Hughlings Jackson and Taylor,[2] and Lépine,[3] in cases of double hemiplegia. The attacks of hemiplegia were peculiar, in that they commenced with comparatively slight paralysis, which afterwards increased. This would seem to have been due to repeated small lesions (small hæmorrhages and thrombosis of small arteries) occurring at intervals in the same neighbourhood, such multiple lesions being evident on microscopic examination in each internal capsule.

In the absence of any reliable history as to the onset of the symptoms, it must always be difficult, or even impossible, to diagnose between double hemiplegia and bulbar paralysis, with paralysis of the limbs (amyotrophic lateral sclerosis). The chief points of divergence seem to be:

(1) That in double hemiplegia the affection of the limbs is not quite symmetrical, but usually distinctly more marked on one side than the other.

(2) The absence of any marked wasting of the tongue in double hemiplegia.

(3) The preservation of electrical excitability of tongue and palate in double hemiplegia, while there may be some diminution of response to both forms of current in bulbar paralysis.

---

[1] *British Medical Journal*, 1877, vol. ii.

[2] *Lancet*, 1892, vol. ii.

[3] *Revue de Médicine*, 1893.

Reviews and Notices of Books.

Grundzuge der physiologischen Psychologie. Von WILHELM WUNDT, Professor an der Universität zu Leipzig. Vierte, umgearbeitete Auflage. Leipzig, Verlag von Wilhelm Engelmann, 1893. Vol. I., pp. xvi., 600. Vol. II., pp. xii., 684 (Index, pp. 649-684), 143 and 94 figures.

Grundriss der Psychologie, auf experimentelle Grundlage dargestellt. Von OSWALD KÜLPE, Privatdocent an der Universität, Leipzig. Leipzig, Verlag von Wilhelm Engelmann, 1893. Pp. viii., 478 (Index, 472-478). Ten figures.

WHEN the "Physiological Psychology" first appeared, in 1874, it was as a single volume, of 870 pages.[1] Lotze and Fechner had already done much to prepare the way for the "new" psychology: but it is hardly an exaggeration to say that the publication of Wundt's encyclopædic work, and the opening of his laboratory in 1879, mark the actual birth of the science. Much of the factual material of this first edition was drawn from the author's already published writings: the *Lehre von der Muskelbewegung* (1858), the *Beiträge zur Theorie der Sinneswahrnehmung* (1862), the *Vorlesungen über Menschen und Thierseele* (1863), the *Lehrbuch der Physiologie* (1865), the *Physikalischen Axiome in ihrer Beziehung zum Causalprincip* (1866), the *Handbuch der medizinischen Physik* (1867), and the *Untersuchung zur Mechanik der Nerven und Nervencentren* (1871). The rest was culled with pain and travail from the most varied sources: Johannes Müller, Fechner, Helmholtz, and a hundred others. The "formative influences" mentioned in the preface are the works of Kant, Darwin, and Herbart.[2]

---

[1] First part published separately, 1873.

[2] This edition was reviewed by Professor Sully in "Mind," O.S., No. I., pp. 20 ff.

The second was a two-volume edition (1880). A French translation of it (Paris, 1886) was issued just before the appearance of the third German edition. French being more generally understood than German, and Wundt's technical German being none of the easiest, this translation was and still is much used and cited. It is, of course, hopelessly antiquated: in fact, fourteen years old, despite its date of publication. The third edition[1] itself (1887) showed a great advance in the progress of the science. So far from its being now difficult to get material together, the difficulty was that of selection from and discrimination within a truly vast literature, written in all the chief languages of Europe. Not only had there arisen the new school of professional psychologists: psychological problems had attracted the attention of scientists and philosophers of every school and interest. But even more formidable than in 1887 was the task of revision in 1893. The fourth edition is, it is needless to say, larger than the third. That, however, is not all; it is very different from it. In the last six years neurology and experimental psychology have changed, as regards fact, method, and theory; and it is attempted to do justice to these changes, in the revised chapters which treat of the two disciplines in question. Moreover, Wundt's own views upon matters of systematic psychology have undergone important modifications, which would of themselves suffice to justify republication of the work, with the title of a new edition.

It must always be a matter of regret to psychologists that Lotze published no review of the "Physiologische Psychologie." Without disrespect to living representatives of the science, it may, I think, be said that he was the one man in every way qualified to undertake the task of criticism and appreciation—having had, like Wundt himself, the preliminary training of a physiologist and physician. It would in oneself be no less than impertinent to attempt a "review," in the sense in which the word has just been employed. The object of the present notice is far more restricted; it is: (1) To furnish those who are already acquainted with the third edition of the work with an outline of the alterations introduced into the text of the fourth; and (2) To give readers who come to the latter, as to an unknown book, some idea of the general contents of it.

The *Introduction*[2] has suffered but little change. In it the author treats, as before, of the problem of physiological psychology and of the presuppositions of psychology in general. A reason is given for the retention of the adjective "physiological" in the title of the work. The expression "experimental psychology" is the equivalent of "psychology," as at present defined. "Physiological psychology" (1) pays more special regard to those psychological investigations whose instruments are physiological than to any others, and (2) discusses the physiological basis of mental life: its field, therefore, is at once narrower and wider than that of "psychology." A sharp criticism of hypnotic "experimentation" refers the reader to the recently published brochure "Hypnotismus and Suggestion."

---

[1] Reviewed by Professor Cattell in "Mind," O.S., No. LI., pp. 435 ff.

[2] 3RD ED., I., 1-20; 4TH ED., I., 1-20. *Omitted:* 3rd Ed., p. 3. *Added:* 4th Ed., pp. 8, 9, *Diejenigen. . . Expanded:* 4th Ed., pp. 4-6. *Condensed:* 3rd Ed., pp. 16 midd.—18.

Part I.[1] deals with the physical basis of mental life, and is mainly taken up with an exposition of the relation of the nervous system to the mind. Chapters and sections are entitled and arranged as in the third edition. The discussion is of immense value, as being the only adequate treatment of the subject extant, from the psychological point of view. Students of neurology, who come to it solely for the purposes of their own science, will probably be disappointed in what they find; it will seem to them that more space should have been devoted to the quite recent discoveries and inferences of nervous physiology. But the attitude would not be a fair one. There is ample indication that Wundt's erudition has kept pace with neurological research, and that, if his physio-psychological position has not greatly changed, this is due to a belief that there is nothing in the many lately-published monographs which calls for its radical alteration.

The chapters contained in this *Part* are six in number: (I.) on the organic development of mental functions; (II.) on the structural elements of the nervous system; (III.) on the gross-anatomical development of the nerve centres; (IV.) on the course of neural conduction paths; (V.) on the physiological functions of the central parts; and (VI.) on the physiological mechanics of nervous substance. Some alterations have naturally been introduced in the second of these;[1] "collaterals" were not thought of in 1887. Indeed, much of the minute work of Ramón y Cajal, Kolliker, Retzius, Golgi; of the gross work of Edinger; and of the functional investigation of Mosso and Horsley is quite recent.[2] In the fourth, the section on the fillet (laqueus) has been rewritten; the twofold nature of the optic and acoustic nerves (as at once centripetal and centrifugal) is discussed; and the probable indifference of function of commissural fibres in the lower central parts remarked. Portions of the section on paths of conduction to the cerebral cortex have been remodelled, though its general conclusion remains as before. The schematic figures of the lines of conduction through *pons* and *cerebellum*, and in *pons*, *cerebellum*, and *cerebrum*, have been brought up to date; but, unfortunately, at the same time reduced in size. The section on sleep (chapter v.; automatic functions: *cf.* chapter xix.) has been revised. Recent experiments on the total extirpation of both hemispheres, in dogs and birds, have led to modification of the section on cerebral function. In the theory of central innervation (chapter vi.), more stress is laid than was previously the case upon the nutritive influence of the central substance on the fibres connected with it. Finally, the schematic and hypothetical representation of the apperception-organ has been changed for the better, and the textual consideration of it to some extent altered. The whole discussion remains modestly in the small print, to which it was relegated in the third edition. One could have pardoned the author, if persistent misunderstanding of his theory had led him to restore it to its original type.

---

[1] 3rd Ed., I., 21-288; 4th Ed., I., 21-280. *Omitted:* 3rd. Ed., pp. 64, last sent. of § 1; 80 note; 87 last sent. of midd. §; 115 last sent. of §; 115 *Ebenso* to end of small pt.; 162 last sent.; 198 *Da nun alle* to 199 end of §; 215 large pt.; 217 second §; 250 last half of last sent. of § 1; 266 small pt. to 269 top; figg. on pp. 30, 72, 74, 75, 84, 86, 98. *Added:* 4th Ed., pp. 34 top to midd.; 91 small pt.; 98 last sent.; 188, 9 *Wahrscheinlich* . . . . .; 166 last sent.; 244 two sents. in middle §; 274 *Aus diesen* . . . . .; 278 last sent. of § 1; figures on 34, 130. *Expanded:* 4th Ed., pp. 38, 9; 127; 130; 192; 201 note; 211 note; 214 second §; *Condensed:* 3rd Ed., pp. 36; 66, 7; 68; 91 note; 111 last part; 166 end of small pt.; 194 last half; 211-3; 231-4. *Modified:* 4th Ed., pp. 81, end of § 2; 146; 188 last sent. of § 1; 228 end; 280, 1; figg. on pp. 122, 141.

Part II.[3] is concerned with sensation. The chapters are headed as in the previous edition: (VII.) the origin and general properties of sensation; (VIII.) the intensity, and (IX.) quality of sensation; and (X.) the affective tone of sensation. But this is one of the two *Parts* which at first sight show the traces of revision most clearly. Indeed, Wundt tells us in the *Preface* that such an impression was intended. Experimental psychology is to-day an independent science; it has its own methods and its own instruments,—methods and instruments as peculiar to it as are those of chemistry to that discipline. It seemed well, therefore, to incorporate a somewhat detailed account of these in the "Physiological Psychology." And, since sensation has proved to be one of the most fruitful departments of experimental psychological research,[1] we find technique and procedure discussed here far more exhaustively than in the book of 1887.

---

[1] The phrase "unipolar cell," despite Golgi's "monopolar" is surely a survival from the older neurology.

[2] Lenhossek, Van Gehuchten, Obersteiner and Cunningham do not appear to be mentioned.

[3] 3rd Ed., I., 289-544; 4th Ed., I., 281-600. *Added:* 4th Ed., pp. 286 top; 293 last sent. of § 1; 295 last sent. of § 1; 303 *Fur die Temperaturreize* . . .; 310 last sent. of § 1; 323 *In der That* . . .; 333 after italics in § 3; 356 ff.; 361-5; 389 ff.; 394; 396 last sent. of § 2; 417 last § of large pt.; 455 midd. §; 462 to sents. at top; 464 *Stosstöne*; 494 top sent.; 509 *Uebrigens* . . . ; 514, 5 *Die günstigen* . . .; 517 *Alle diese* . . .; 520 last word to end of § on 521; 529 midd. §; 542-4; 549 §; 550 midd. §; 558 § 2 to 563 top, much added; 582-6; 596 to end; figg. on 300, 308, 315, 361, 362, 363, 373, 374, 409, 459, 502, 506, 542, 543. *Omitted:* 3rd Ed., pp. 328, *Da aber* . . .; 334 last sent. of § 1; 348 *Dabei ergänzen* . . .; 349 § 2; 359 last sent. of § 1; 441 last sent. of § 1; 578 last sent. of small pt.; 580 top 6 lines; figg. en 300, 305, 315, 324, 327. *Expanded:* 4th Ed., pp. 297, last sent. of § 1; 306, 7 *Von den* . . .; 314, 5; 319, 20 *Trotz* . . .; 327 top; 328, 9; 329 to midd. of 331; 342-58; 359, 60 sent; 360, 1; 372 ff.; 381 ff.; 383 ff.: 387 ff.; 396 last sent.; 404, 5; 410 to 414 top; 417-9; 419-22 top; 422 midd; 422-6; 426-33; 440, 1; 450, 1; 464, 5, 6; 466; 468, 9; 472, 3; 473-81; 500-3; 505, 6; 515, 6; 521 midd. §;

I have said that Wundt is the father of experimental psychology. The younger generation of laboratory-trained psychologists is divided, as things are, into two groups: an orthodox school, which accepts Wundt's cardinal doctrine—that sensation is not the sole elementary mental process, but that conation is as radical as sensation chronologically, and prior to it logically; and a school of dissent, which denies primitiveness to any other constituent of mind, explains affection (pleasure-pain) and conation in terms of sensation, and seeks to affiliate experimental results to the theories of the older English associationist psychology. Both parties point to experience as their witness. The former says: We find in consciousness we experience, a conative process, which is definite as regards quality, intensity, and duration. The latter: We find only sensation-processes; an idea of

---

536; 552 3 last sents. *Condensed:* 3rd Ed., pp. 291, 2; 321, 2; 361; 365; 380, 1; 388, 9; 395; 421 midd. §; 426 *Zugleich* . . ., 432 § 2; 433; 470, 1; 490; 499, 500; 503; 513 to top of 515; 543, 4. *Modified:* 4th Ed, pp. 281; 282; 287; 299 last §; 327 § 2; 366, 7; 370 fig.; 370, 1; 438-40; 448 top; 468 end of § 1; 488; 500 end of § 1; 523 midd. §; 526 end of § 1, 529 top.

[1] I should like to take this opportunity of protesting against the widely spread and frequently expressed view, that experimental psychology can never deal with the "higher" mental processes. "Experimental psychology," as Wundt puts it, is just "psychology," the impossibility of experiment is as wide as mind. To answer the objection more concretely: (1) the sensation is the easiest psychological material to work on, and therefore has, naturally, been taken first; (2) the doctrine of sensation is by no manner of means completed so that, in assigning subjects of investigation to comparatively unskilled students, directors of laboratories tend to choose them from within its limits; (3) the statement is disproved by facts; fusions of such a high order as attention, emotion, and apperceptive ideational combination having been experimentally investigated under various aspects. Experimental psychology simply needs time and trained workers. Any restriction of the phrase such as that which the word "psycho-physics" has undergone is to be deprecated; *cf.* the remark made at the conclusion of the notice of Dr. Kulpe's *Grundriss*, below.

movement is to us the will to move, strain-sensation complexes are attention. And experience must be the final appeal: it is only a matter of better or worse introspective analysis. No one recognises this more explicitly than Wundt himself, whose theory of conation or apperception is a strictly empirical, experiential theory—as "unmetaphysical" as most current hypotheses in the natural sciences are "metaphysical." Sensations, therefore, are defined, at the outset of this *Part*, not as the simplest elements of consciousness, but as the simplest elements of ideas: the idea being further described in Vol. II., as the mental image of an object or process of the external world.

There is but little alteration in chapter vii. A new paragraph deals with the terminal apparatus of the temperature sense. The treatment of the ear is modified to correspond with alterations in the treatment of the auditory idea in *Part III.*; that of the eye to correspond with the altered *opticus* schema in *Part I.*—a schema, by the way, which is here turned to account for the explanation of the "projection" of hallucinations and fancy-images. The critical and historical discussion of the doctrine of specific energies is much fuller than in the previous edition.

The detailed account of the psycho-physical measurement-methods (chapter ix.), on the other hand, is greatly enlarged. These methods are, of course, employed with the object of obtaining exact knowledge about mental processes, considered under various aspects. But their elaboration and formulation have proved to be so difficult, that there has arisen a very considerable literature, devoted to the interests (mathematical and other) of the methods themselves, without regard to the conscious content for whose examination they were primarily designed. This literature is not psychology, but it is useful to the psychologist: and, so far as that is the case, it is reproduced in Wundt's summary. Much new matter is also introduced in the discussion of Weber's law in the various sense-departments. The summing-up upon the general validity of the law, is longer and more cautiously worded than it was six years ago; but the general verdict is still favourable, despite the complication by proof of the existence, under certain conditions, of a direct proportionality between stimulus and sensation.[1] Similar alterations are noticeable in chapter x. The cutaneous and deeper-lying sensibilities are in particular subjected to a more exhaustive qualitative analysis. "Sensations of innervation" and the "sense of feeling" are finally given up, on account of the liability of the expressions to cause misunderstanding. The exposition of Wundt's own theory of light-sensations, which is the only non-component hypothesis in the field, is very opportunely lengthened and clarified.

---

[1] It is curious that the degrees on the brightness scale (black through grey to white) are treated throughout as though they indicated intensity-differences only, and not quality-differences. See, however, p. 505

The author's attitude to pleasure-pain content remains more conservative than the treatment of it in the second edition of the *Vorlesungen* might have led one to expect. The younger Wundtians are apt to look upon sensation, affection, and conation as equally primitive and equally elemental independent processes.[1] Such a view is not inconsistent with Wundt's theory of affection—that it is the mode of reaction of conation upon sensation; but he prefers himself to speak of the "affective tone of sensation," and to look for the conditions of pleasure or pain in the sensation, rather than the stimulus-process. I cannot here enter upon the reasons which lead many psychologists to a severance of affection from sensation, and to the preference of its correlation with conation. They seem to me to be weighty, and to be implicit—indeed, partly explicit—in Wundt's own system. Chapter x. contains much that is new: in the analysis of the concrete pain-feeling, the added section on the physical correlates of the sense-feelings, the discussion of theories of the origin of these.

Part III.[2] on the formation of ideas, contains four chapters: (XI.) general considerations; ideas of touch, movement, (XII.) ambition, and (XIII.) vision; (XIV.) the elementary æsthetic feelings. The tactual idea and the theory of tactual space are handled at greater length than before, and their mode of treatment modified, just as was the case with the cutaneous and subcutaneous sensations in Vol. I. The phrase "psychic synthesis" (as descriptive of the sum total of mental processes underlying the construction of non-visual space) has been replaced by the less ambiguous "extensive fusion." "Fusion," to Wundt, is still no more than very intimate association. He does not separate fusion (= intensive or qualitative combustion of elements, in which the constituents are overshadowed by the resulting total-process) from association (= temporal or spatial combination, when the constituents are in the result as clear as or clearer than they were in isolation), as, *e.g.*, Külpe does. This is shown in the added pages on tonal fusion in chapter xii.; the other more important alterations in which are the fuller discussion of auditory localisation, and the introduction of new sections on the temporal association of auditory ideas. The next chapter shows the traces of revision most clearly in relation to optical illusions. It is, of course, one of the most thoroughly Wundtian parts of the work. Of the three moderns mentioned by Professor Sully[1] in 1876, as mainly responsible for the "derivative" or "empiristic" theory of psychological space, Helmholtz vitiated much of his reasoning by confusing logic with psychology, while Lotze adopted a nativistic metaphysic, which deprived his "local organs" of their real importance. Wundt's views have been subjected for a quarter of a century to the tests both of internal and external criticism; and are, in their present form, typical of the opinions of one of the two main schools of space-psychology. Chapter XIV. contains new paragraphs, dealing with the relation of the elementary æsthetic feelings to other forms of the feeling-process.

---

[1] Independent, of course, only in abstraction; not in the concrete mentality of the individual.

[2] 4th Ed., II., 1-254; 3rd Ed., II., 1-224. *Added:* 4th Ed., pp. 1 *Beide* . . .; 13 *Dies erklärt* . . .; 19 last sent.; 21 *Hierbei* . . .; 28, 9; 33 *Werden dagegen* . . .; 71 top §; 71-4; 87-9; 95, 6; 100 last sent. of § 1; 122, 3; 129 *Die geschilderten* . . .; 137 midd. §; 142 midd. §; 155, 6; 172, 3 sent.; 245-8; figg. on 123, 129, 146 ff., 201, 241. *Omitted:* 3rd Ed., pp. 3 *Nun haben* . . .; 45 top sent.; 72 3 sent.; 80 last § of small pt.; 198 top sent.; 207 *Ebenso* . . . *Expanded:* 4th Ed., pp. 27, 8; 29-32; 50, 1; 60; 66, last §; 94, 5; 133; 139, 40; 146-52; 240, 1. *Condensed:* 3rd Ed., pp. 11, 12; 21-3; 24 *Wenn man* . . .; 54 second §; 80 1st two §§; 115 midd. §. *Modified:* 4th Ed., p. 23; 26; 38; 41; 83; 101, 2; 140-2; 142, 3.

Part IV.,[2] of consciousness and the train of ideas, is the other of the two *Parts* which show the most obvious and unmistakable evidence of revision in the new edition. Its chapters treat (xiv.) of consciousness; (xvi.) of apperception and the train of ideas; (xvii.) of ideational combinations; (xviii.) of emotional phenomena; and (xix.) of disturbances of consciousness. To be noted at once are certain alterations in systematic psychology. In the first place, the process of apperception is more clearly separated out from its conditions and associated processes; those determinants of a train of ideas which are to be looked for in the general psycho-physical trend or disposition of an individual at a given time, being now definitely marked off from the conative elements in the whole mental content of that time. The attention is regarded, in essential, as a phenomenon of inhibition (chapters xv., xvii). Secondly, time-ideas (*cf.* chapter xii.) are discussed in a new section, principally upon the basis of Meumann's recent investigations; extant theories of the "time-sense" are criticised; and the form which an adequate theory must take is pointed out. The psychology of time is not, of course, established upon so secure a basis as that of space; but light, factual and methodological, is beginning to break in upon us. I hardly know of any series of special researches, the following out of which shows more distinctly the difficulties and special fallacies that beset the path of the psychological experimenter, than that dealing with the "time-sense," which begins with Vierordt and Mach, and ends with Schumann and Meumann. Thirdly, the doctrine of association of ideas has been very thoroughly revised; a long section added, on the theory of association; and, as indicated above, the apperceptive factors sharply distinguished from the associative. An adequate discussion of any one of these three changes would in itself demand several printed pages. Here they can only be barely mentioned, with the remark that they are all in the direction of true psychological progress, and that there is a complete absence of indication of that fossilisation of thought, which maturity is so apt to bring to speculative and constructive minds. No one who has had the privilege of personal intercourse with Wundt can have failed to be impressed by his openness to new ideas.

---

[1] "Mind," O. S., I., p. 24.

[2] 3rd Ed., II., 225-462; 4th Ed., II., 255-559. *Added:* 4th Ed., pp. 276, 7; 291, 2; 294 last sent.; 311 note; 315-7; 318 second table and remarks to 319 top; 346 *Martius*; 357, 8; 379, 80; 429-31; 436; 441-447; 480 *In der Ausbildung* . . .; 480-2; 497-501; 508 *Wird ein Streben* . . .; 546 *In der Neigung* . . .; figg. on 291, 359, etc. *Omitted:* 3rd Ed., pp. 258 last sent. of § 1; 271 *Zu einem grossen* . . .; 291 last sent. of long §; 410 two last sents. of top §; fig. on 297. *Expanded:* 4th Ed., pp. 266-8; 271-5; 275, 6; 279-82; 282-6; 295-300; 322-44; 344, 5; 347, 8; 360-2; 363 long §, 420-9; 431 § 1; 457-64; 466-75; 543; 545, 6; 547 midd. §; 552-4. *Condensed:* 3rd Ed., pp. 240-2; 244, 5; 285, 6; 409 midd. §; 438-40; 453, 4, 456-9. *Modified:* 4th Ed., pp. 266; 300, 1; 348, 9; 370 midd.; 408; 408-20; 431.

Attention may be called to some few of the other alterations of *Part IV.* In the first place, the technique of mental chronometry—a department of investigation in which Wundt's first experiments date back to 1860—and of the "time-sense" experimentation, is elaborately described; figures of apparatus given; and typical sources of error pointed out. The recent confirmations of the view that the so-called "fluctuations of attention" are really what their name states them to be, and the present state of the controversy, as regards the two fundamental forms of the simple reaction, are set forth fully and clearly. The experimental basis of association-theories is seen to be still very inadequate, though the most is made of the little work that has been done. An entire section is added in chapter xviii., on the general interconnexion of affective processes. For most of these changes, readers of the *Philosophische Studien* will have been to some extent prepared.

Part V.[1]—on will and the external voluntary action—has remained practically untouched. It contains chapters (xx.) on the will; (xxi.) on the influence of the will upon bodily movement (automatic and reflex; impulsive and voluntary); and (xxii.) on expression of emotion. I have already said that the doctrine of conation is the hinge upon which the whole of the Wundtian psychology turns. For a complete understanding of this doctrine, the present chapters should be read in connection with parts of the *Ethik* and *System der Philosophie*, with certain of the *Essays*, and with various articles in the *Phil. Studien.* The titular limitation of the work under consideration would of itself indicate the necessity of such comparative reading.[2]

And it has, naturally, been found equally unnecessary to change the exposition of *Part VI.*,[3] which treats in two chapters—(xxiii.) metaphysical hypotheses as to the nature of mind; (xxiv.) general outlines of a theory of our inner experience—of the origin of mutual development.

If Wundt has spared no pains to make "physiological psychology" complete, within its limits, he has himself called our attention to the fact that these limits are not so wide as those of the science of "psychology." Indeed, as I have pointed out above, this would have been a necessary inference from the scheme and plan of the work. After a brief introduction to the general subject, we are shown in detail the bodily structures and functions with which mentality is correlated. We then enter upon the study of the most objective of simple mental processes—sensation; which is handled in both general and special physiological reference. In the third place, we examine the ideas of sense, so far as this is possible under the assumption that they are simply groups or complexes of sensations, *i.e.*, omitting for the time being the consideration of the conative factor in their composition. Up to this point the task of correlation has been, in theory at least, fairly easy. But now our faculty of analysis is to be more severely tasked. We are taken to the concrete mind; its "states" of attention and emotion, its "trains" of ideas, and the abnormal conditions into which it is liable to fall. Here the physiological processes co-ordinated with the mental are of a higher order of complication and less exactly known: care is needed, lest the investigator, anxious to make his parallel complete, fall into a barren schematism, which shall tend to hinder the later acquisition of real knowledge. Lastly, and with the greatest difficulty, we approach—always with the physiological reference—the concrete study of that side of mind which we have learned to know in its simpler form in the "states" and "trains" already handled—the will. And a final section co-ordinates the different views which we have been led to adopt as the investigation proceeded, and shows us the justification of the assumptions which we made, as working hypotheses, at the outset of our inquiry.

---

[1] 3rd Ed., II., 463-530, 4th Ed., II., 560-625. *Omitted:* 3rd Ed., pp. 480 last. sent of § 1; 480 *Eben darum* . . .; 482 last sent. of midd §; 486 *Von psych. Seite* . . ., 489 last part of §; *Condensed:* 3rd Ed., pp. 483, 4. *Modified* · 4th Ed., pp. 575 ff., title.

[2] In the same way we shall go to the *Logik*, in order to supplement the psychological doctrine of the "Phys. Psych." on other sides.

[3] 3rd Ed., II., 531-554, 4th Ed., II., 626-948. *Omitted.* 3rd., Ed., p. 544, last sent. of § 1. These footnotes will have shown what it is hardly needful to mention, that a very large number of textual modifications has been entirely passed over in the above notice. Some of them are of considerable psychological importance, *e.g.*, the analysis of sense-pain.

The programme is coherent and complete in itself: but, to quote from Wundt again, it contains both more and less than would the programme of "psychology." We require from the author another special psychology: a "social psychology," which shall deal with myth, language, and custom. The lectures on this subject, which have been delivered for several years, will surely one day appear in book form. May we not also require from him a systematic psychology,[1] freed from *any* such definite reference? An example has already been set by Professor Ladd, who publishes a "Psychology" before a second edition of his "Physiological Psychology" (1887) has been called for.

The two volumes are well printed and have good margins; the paper—*O si sic omnes!*—is a deep cream, and not white;[2] the cuts are clear, and better set than in the third edition. Dr. Meumann has furnished an excellent index of matter. Is it ungenerous to end with the wish that as good an index of names had also been provided?

Dr. Külpe's book is dedicated to Wundt, and is throughout influenced by Wundtian modes of thought, the other influence most distinctly traceable being that of G. E. Müller. It is undoubtedly the best text-book of experimental psychology extant; and—what is not necessarily implied in that statement—it is a thoroughly good text-book, regarded on its own merits. The value of its parts, from this point of view, is of course very different. Some chapters are based upon an extensive experimental literature; others, the experimental material for which is still scanty or untrustworthy, the author has been obliged to construct as best he could from these scanty materials, and to elaborate by the aid of hypotheses erected upon admittedly insecure foundations. But it is no small thing to have written the first adequate manual of the science. And the high reputation which Dr. Külpe had already won by his published articles will certainly be increased by the quality of the present book.

---

[1] *Cf.* J. W. "Mind," N.S., No. 9, p. 143.

[2] If one is to judge from present indications, it will still be long before publishers and public recognise the fact that there are a real right and wrong about the make of a book: its size, type, tint of paper, &c. The glaring white of so much note paper and of so many *éditions de luxe* is hopelessly "unpsychological."

I can only note the contents and its arrangement in the briefest way. The *Introduction* (pp. 1-29) deals with the problem of psychology, its methods and the assistance which it receives from related disciplines, and with psychological classification and literature. *Part I.*, on the elements of consciousness (pp. 30-283), falls into two sections, entitled respectively "Sensations" and "Feelings." Prefixed to the detailed consideration of the properties of sensation is a chapter (1) on sensational analysis, which, in pædagogical reference, must be termed no less than masterly. The author's long experience as assistant in Wundt's Institute has given him a familiarity with the use of the psycho-physical measurement-methods, and an insight into the difficulties presented to the beginner in the study of them, which have combined to make his exposition a model of conciseness and lucidity. The following chapters treat of (2) the quality and (3) intensity of peripherally excited sensations; and, under the title "centrally excited sensations," of (4) reproduction and association. The discussion of the latter processes is more than a text-book *résumé*: it is a real and independent contribution to systematic psychology. For that very reason, however, it does not stand upon the plane of the previous chapters; its conclusions are, from the nature of the case, more individual and debatable. In the section on feeling are included paragraphs dealing with the question of an elementary conative quality.

Part II. (pp. 284-437), on the combinations of the conscious elements, falls similarly into two subsections; on "Fusion" and on "Association." I have above called attention to the difference between these two forms of combination, and think, with the writer, that they should be separately handled, and that separate theories are required for their explanation. The fusions discussed are (1) those of tone sensations, (2) of brightness with colour, &c., and (3) of sensation with feeling (impulse, emotion). Under the association title we have chapters on (1) the spatial properties and relations of sensations; (2) the temporal properties and relations of the elements of mind; and (3) temporal and spatial association. Cases of the latter are visual contrast, and the simple reaction (= the exact type of an "action.") The "association of ideas" is not further treated of (*cf.* *Part I.*, ch. 4).

Finally, the "state" of consciousness forms the topic of *Part III.* (pp. 438-471). "States" of mind are attention, voluntary thought, self-consciousness, dreaming, hypnosis, &c.

The author professes himself a disciple of Wundt, as regards the latter's doctrine of apperception. But, ingeniously as he reconciles his own view with Wundt's terminology, and dexterously as he takes advantage of Wundt's latest modifications of his theory of attention, the fact cannot be overlooked that the standpoints of the two psychologists are at bottom different. Effort—the quality of will—is reduced to a complex of sensation qualities (p. 275). There is no special conscious content, "internal activity" (p. 440). But this is hardly a restatement of the teaching of the "*Physiological Psychology*" II., 266, ff., to say nothing of other passages in Wundt's writings. A feeling of loyalty may have prompted Dr. Külpe to minimise his divergence from his teacher: but the difference is certainly there.

I do not intend, however, to give here a detailed criticism of the work. Only one remark may be made in conclusion: that the arrangement of the book does not seem to be the best possible. To treat of emotion and impulse before attention has been touched is, I think, bad pædagogical policy, even if the order of treatment do not rest upon faulty psychological analysis. The elementary will-quality is out of place among the feelings. Expressions of the emotions should be dealt with under the heading of "association" (*cf.* p. 343). The association of ideas might well have been spared a few more pages—again for pædagogical purposes—in the chapter discussing contrast and action. That the author is upon the track of the right arrangement of an experimental psychology, I have little doubt, but I incline to think that a fear of over-schematisation has led him to depart from the logical consequences of his original plan.

Find fault as one may, however—because the contents are not ideally arranged, because the book is part manual, part constructive psychology, because it is so predominantly German in reference and appreciation—the fact remains, as stated at the outset, that Dr. Külpe has produced a thoroughly good text-book of experimental psychology. It is none the less welcome, as a complete disproof of the notion that the science has covered no other ground than that of sensation.

E. B. TITCHENER.

Métaphysique et Psychologie. Par TH. FLOURNOY, M.D. 8vo. pp. vii., 133. H. Georg. Genève. 1890.

This little work, which appears to be an amplified edition, with notes, of the second of two "conferences," or lectures, on "Soul and Body," delivered in certain public courses under the auspices of the University of Geneva, in December, 1888, is an explanation and defence of the place which physiological psychology has won for itself, during the last thirty or forty years, among the positive and experimental sciences. The sitting at which it was pronounced was devoted, we are told (p. 52), "to determine the line of demarcation between positive psychology and metaphysic," a task of great interest, not for amateurs only, as the author modestly suggests, but also for those whose main business lies either in scientific or in philosophical pursuits, and moreover a task for which the lecture itself shows, that its author is eminently qualified.

Nothing can be clearer than the exposition which M. Flournoy gives of the way in which physiological psychology has become detached, as it were, from the philosophical group of sciences (so to call them), has asserted its autonomy, and has taken rank as the youngest of the positive and experimental sciences, a science possessing its laboratories and its instruments of precision, and proceeding to reduce its phenomena to measurement, which is the essential mark of all exact science. Nothing can be more definite than the author's statement of the two great principles, upon which, as he holds, this position is based, and by which it is justified. These principles are, first, the principle of concomitance or parallelism: "Every psychical phenomenon has a determinate physical concomitant" (p. 5); and secondly, the principle of dualism, or axiom of heterogeneity: "Body and mind, consciousness and molecular cerebral movement, the psychical fact and the physical fact, although they are simultaneous, are heterogeneous, disparate, irreducible [to one another], obstinately TWO" (p. 17).

I will endeavour to reproduce in English the paragraphs which lead up to this definition as their conclusion, seeing that they set the author's fundamental view in the clearest light: "How can two things so opposite as mind and matter, the physical and the moral, find themselves in relation and influence each other? This is what no one has yet explained, and the most subtile thinkers have spent in vain all the resources of their genius in trying to solve this irritating problem. Let philosophy at the present day go on fathoming it—that is its business. But if psychology yielded to this temptation, all progress would for it be at an end. Psychology cannot advance, cannot even subsist as a science, save on condition of going straight on, without troubling itself with this mystery, any more than if it did not exist. Yet we are aware that it is no easy matter thus all at once to ignore a sigh of interrogation, which thrusts itself forward at every step. It is a ply to take; time is requisite. Accordingly it is well that, until the habit is acquired, the newly-born science should find in its fundamental principle itself a perpetual recall to order, and the warning that it has resolutely to turn its back on metaphysic.

"This warning is already contained in the very terms, *concomitance, accompaniment, parallelism,* and other equivalents, often employed to designate the principle in question. Just as two parallel lines, or two persons who are walking together, continue distinct and separate, and go on moving in company, without fusion and without collision, so, in the eye of experimental psychology, the series of psychical facts and the series of physiological events, while flowing on side by side, remain always different, distinct, without points of contact, without any conceivable mutual influence. They are simultaneous, that is to say, have a time-relation to each other, but that is all — no discoverable relation of action or of nature—they are heterogeneous.

"It is advisable, in order the better to bring out into prominence the important corollary of the principle of parallelism, to formulate it separately, as an independent truth, under the name of Axiom of Heterogeneity or Principle of Dualism." Then follows the definition which has been quoted above (pp. 16-17).

The first chapter having thus been devoted to set forth the essential principles of positive psychology, our author proceeds in chapter ii. to criticise the main classes of what he calls the metaphysical hypotheses, which have been propounded concerning the nature of the union between soul and body. These he reduces to four, namely, spiritualism, materialism, phenomenism or idealism, and monism. With these, however, and with his highly instructive criticism upon them, it is not here the place to concern ourselves. I propose similarly to pass over the discussion, with which his third and last chapter is occupied, on the position which science generally, and physiological psychology more particularly, hold with regard to the problems, first, of a future life, secondly, of free will and moral responsibility. Of the first he says: "Its account is soon cast up—nothing for it, nothing against it" (p. 53). As to the second, his conclusion is, that, while no positive science can even entertain the question of liberty, determinism being its necessary pre-supposition as science, yet, seeing that determinism is not necessarily "the expression of absolute truth," it is beyond the true province of science either to affirm or to deny it, and that the attempt to do either the one or the other would at once rob science of its independence, and change it *pro tanto* into dogmatic philosophy. It is in short (in his view) a metaphysical and not a scientific question (pp. 83-84).

The rest of the book is occupied by notes, the greater part of which relates to the great principles expounded in chapter i. It is here that the main interest of the work is centred, at least for neurologists. It is the view taken of the constitutive principles of physiological psychology which determines what we are to think of the other matters which the book contains, or in other words, what relation we are to conceive psychology holding to the four so-called metaphysical hypotheses criticised in chapter ii. and to the questions of a future life and free-will discussed in chapter iii. It is, moreover, those constitutive principles of the new science, physiological psychology, which have most interest for men of science, and especially for neurologists, inasmuch as they determine its relation to its own better half, if so it may be called, namely, physiology. The following remarks will accordingly be addressed to the consideration of these two cardinal or constitutive principles.

And here the first and most prominent thing which strikes us is the author's open and repeatedly expressed dislike and contempt for metaphysic, founded, as will presently appear, on a misconception of what metaphysic really is. His attitude towards it is one of undisguised hostility. He speaks of "shaking off the yoke of metaphysic, by banishing from science the age-long quarrels of philosophers" (p. 6); he quotes against it a well-worn joke as to its utter unintelligibility (p. 7); every metaphysical system, he says, is well-known to have a false pass-key to the gate which opens on the Absolute (p. 20); he would give them all their final *congé* (p. 51); he speaks of the serene indifference of science in matters of philosophical belief (p. 53); metaphysic, he says, gives everyone exactly the answer he wishes, in a certain important question (p. 99); and, at p. 116, "We gladly make allowance for jugglery (*escamotage*) in persons who get their living by it, for instance in *la Métaphysique*, whom the force of circumstances has always reduced to this extremity." Who can think that any one is likely to draw the line of demarcation truly between pyschology and metaphysic, whose views of the latter are of this nature? If mataphysic is jugglery, what need of 133 pages to demarcate it from science?

It is not to be supposed, that this feeling of hostility is anything like universal in the scientific world. It is probable that many men of science would gladly see an alliance between positive science and metaphysic, provided of course that a metaphysical system should be found possible, which is at once solidly based on facts of experience, has a method and principles of its own, and neither pretends nor attempts to impose *a priori* dogmas on any positive science. A metaphysical theory of this kind would probably be welcomed by many a man of science, as serving to complete the system of the positive sciences by showing what are the nature and limits of all speculative thought, upon whatever part of the field of knowledge it may be directed, including those of Ethic, Logic, Æsthetic, and Theology. Some further remarks on this subject will be made, in conclusion of the present article.

Now the establishment and recognition of physiological psychology as a positive and experimental science is, to say the least, as great a benefit to metaphysic, in the true sense of that word, as it is to psychology. It is at least as much the interest of metaphysic as it is of psychology, that the line of demarcation between them should be truly drawn. Metaphysic as much as psychology will be a gainer, by being definitely relieved of questions which do not properly belong to it, and which, while undistinguished and uneliminated, have been a perpetual source of confusion and embarrassment. We may therefore heartily concur with M. Flournoy in his wish to establish the true line of demarcation, though not concurring in his hostility to metaphysic. We may think with him, that it is the interest of science to be relieved of metaphysical questions; but we may think so for the additional reason that the metaphysician in every man, even in men of science, will thereby be most surely appeased and satisfied.

The whole question as to what the true line of demarcation is turns upon the sufficiency of the two great principles which M. Flournoy has laid down, concomitance and heterogeneity, first to constitute a positive science, secondly to keep it clear of all metaphysical matter. The constitution of psychology as a positive science on these two principles is M. Flournoy's main theme. The sufficiency of those principles for the establishment of a science free from metaphysic is therefore the cardinal point to which our attention is directed. To the present writer it seems that their insufficiency is manifest, and that for both the purposes which they have in view. They neither suffice to constitute a positive science, nor do they exclude metaphysical conceptions, in M. Flournoy's sense of the term, from the science which they would constitute.

Let us begin with the principle of concomitance. By this is meant, as M. Flournoy repeatedly explains, that, between the two heterogeneous components, which together make up the total subject-matter of psychology—the neuro-cerebral process and the process of consciousness—no relation save that of simultaneity can be admitted by psychology to exist. Wherever there is consciousness, there some phenomena belonging to these two processes are found accompanying each other, in a regular and constantly recurring manner, and that is all that we know or can know about them; beyond this, whatever we may imagine of their relations is metaphysic.

Now to this narrow idea of what is requisite to constitute a positive science there is a most serious objection. Granting that the two series, the events of consciousness and those of certain neuro-cerebral processes, are totally heterogeneous, and their respective parts strictly and regularly simultaneous, this fact of simultaneity, concomitance, or parallelism, is not alone enough to weld them into the subject matter of a single science. Simultaneity roughly observed may have been the preliminary step, the first incentive, towards a science; but the science is not constituted until the relation of dependence is added to and combined with that of mere concomitance. It is the dependence of one phenomenon on another, whether reciprocal or otherwise, that all positive science seeks to establish. The idea of dependence, or of real conditioning of one thing by another, is the root idea in which science itself, as a mode of knowledge, takes its origin. Let us hear M. Flournoy himself on this point: "What is it, in fact, to *know* an event, to understand it, to make it an object of science, but to connect it with its causes, that is to say, to assign the series and the complete grouping (*l'ensemble*) of antecedent events which have produced it, which have rendered it necessary? To explain a fact is always to refer it to other facts in which it was implicitly contained, and in virtue of which it could not but be, it could not be otherwise than it is" (p. 64.)

In point of fact, the very same observations which show the concomitance of the two series of events, physiological and psychical, show also the dependence of the psychical on the physiological, in respect of their coming into and continuing in existence. In natural or rather in non-artifically instituted processes, as for instance in external sensation, we find the sensation dependent upon, as well as concomitant with, neural processes which are set up directly by the physical environment; we never find sensations setting up the neural processes which are concomitant with them. Take away the neural process, and there is no sensation. Take away the sensation—it cannot be done, save by taking away the neural process. There is therefore dependence of the sensation on the concomitant neural process, but not *vice versâ.*

The same reasoning must be taken to hold good also of intracerebral processes and their concomitant psychical events or states of consciousness, I mean such as association, thought, volition, and emotion; though since here we have not the external agencies, which directly set up neural motion, to appeal to, the demonstration of the fact is far more difficult, and the anticipation of it is still awaiting its complete experimental justification. Its truth, however, is not precisely what I am now concerned to prove. I assume, with M. Flournoy, the thorough-going concomitance of the two sets of phenomena, and then I say, that the same reasoning, which justifies the assumption of concomitance, also justifies and renders necessary the assumption of dependence.

Again, in artificially instituted processes or experiments, we have no other means of laying hold of, producing, or so to speak bringing into court, states of consciousness, save by setting up physical changes which act upon the neural or neuro-cerebral system. Here again, whenever we have reason to infer concomitance, there we have reason to infer dependence also. Indeed it is only by means of the dependence that the idea of a precise concomitance is arrived at; seeing that exact simultaneity, that is, the precise coincidence of beginning and end, between a psychical event and the neural process which it accompanies, is matter of supposition and definition, and not a fact which is capable of being directly observed. By dependence I mean of course dependence of psychical phenomena on physiological, but not *vice versâ.* There is thus no fear of our being led to suppose "a mixed chain in which purely psychical events (sensations, desires, volitions, &c.) should come to be intercalated between physical events (centripetal or centrifugal innervations)" (p. 86). Of the two heterogeneous and concomitant series, the psychical is dependent on, as well as concomitant with, the physiological.

And what is more, this fact seems to be tacitly assumed and counted on throughout M. Flournoy's book, though in express words he repudiates it.

The main reason which he alleges for repudiating it is found in his second constitutive principle, the principle or axiom of heterogeneity. "Since it is just the existence of a bond (*lien*) between two things so heterogeneous which is incomprehensible for our intelligence, it is better to make no allusion to it" (p. 86). But the existence of a bond between physical events taken alone (or for that matter, between psychical events taken alone), is just as incomprehensible for our intelligence, as one between physical events and psychical. Cohesion, attraction, chemical affinity, for instance, are in their ultimate ground or reason equally beyond our power to conceive; the so-called bond between the facts or events which we refer to them is equally incomprehensible; and why? Because, taken alone, it is a name hypostasised and nothing more; no bond other than what is included in the facts or events themselves exists. The terms just cited are names for facts, for events, for phenomena connected in time and space, which we either perceive, or else infer from perceptions, in cases which are beyond the range of our direct perceptive power; and similarly the connection between physical and psychical events is an observed fact, the ultimate ground or reason of which we cannot penetrate. But we do not hesitate to describe physical events as dependent one on another. In their case the *fact* of their dependence suffices us. So also it should suffice in the case of psychology. The heterogeneity of the phenomena between which it is observed adds nothing to the supposed incomprehensibility of its ultimate ground or reason; for that incomprehensibility is in all cases alike at a maximum.

If it should be replied, that after all what we really know of dependence is reducible to the co-existence or succession of the phenomena concerned in time and space, and that this is what is meant by calling it *concomitance* and nothing more, the answer is, that this is equally and alike true of physical phenomena taken alone; that notwithstanding this, we can and do distinguish between cases of dependence and cases of mere simultaneity, or concomitance, without dependence; and that the establishment of the fact and laws of dependence is the sole purpose of science. To science the fact of mere simultaneity is always an *explicandum*, and not an *explicatio*.

A simple and well-worn instance will make this clear. As the earth revolves on its axis in presence of the sun, it turns opposite segments of its surface successively to the sun's light and heat. As one segment of its surface comes into the light of day, the opposite segment comes into the darkness of night, and so on perpetually. The two phenomena, of one segment coming into one position and the opposite segment coming into the other position, are strictly concomitant or simultaneous, but there is no dependence of either on the other. On the contrary, both alike depend upon the physical fact (which is also concomitant with the whole succession) of the revolution of the earth on its axis in presence of the sun. That is the fact which it has been imperative on the science of astronomy to ascertain.

But this does not exhaust the instructiveness of this simple instance. It deserves dwelling on a little longer. The light of day and the darkness of night are sensations. They belong to M. Flournoy's psychical series of phenomena, as distinguished from his physiological series. As sensations there exists between them the relation of contrast. As sensations of a simple individual, there is between them the relation of successive alternation in time-sequence. As sensations thought of, by the psychologist, as being experienced by differently placed individuals, they stand in the relation of simultaneity. Now what has all this to do with the interests of psychology? Simply this, that it is something for psychology to explain. And how? Why by ascertaining, first, the dependence of the sensations in question on states of the neuro-cerebral organism, and secondly, the dependence of these states on the ethereal vibrations with which it comes into contact. M. Flournoy seems to suppose, that there can be no dependence of one phenomenon on another, unless the phenomenon called dependent reacts upon that on which it depends. But for this notion, in the case of psychology, there does not seem to be the smallest warrant. All the ascertained facts indicate dependence (as well as concomitance) of psychical on physiological phenomena, without a trace of any reaction of the psychical on the physiological.

What, then, it may be asked, do you precisely mean by the dependence of some phenomena on others? The answer is—dependence means a relation such that, given the conditioning phenomenon, the dependent phenomenon invariably occurs, and in the absence of the conditioning phenomenon, the phenomenon called dependent invariably makes default. The idea that, to establish the fact of dependence between phenomena, we must have a knowledge of what constitutes a bond or *lien* between them, is an idea which has long been given up in the physical sciences. To retain it, as M. Flournoy does, in the new science of physiological psychology is retrograde, and not retrograde only —it is suicidal.

For observe, in basing psychology on the mere concomitance of its two sets of heterogeneous phenomena, excluding the fact of the dependence of the psychical on the physiological, M. Flournoy not only narrows its foundation, in contravention of positively observed facts, but he robs it of its rank as a positive science. Previous to the rise of physiological psychology, there were two claimants in the field for the office of being the really conditioning agent or agency in generating, sustaining and governing the phenomena of consciousness. One was the soul, mind, or spirit; the other was the ego or some other form or mode of consciousness itself. For, as remarked above, all science aims at ascertaining the laws of the real dependence or inter-dependence of phenomena. The value of physiological psychology consists, therefore, in the fact that the neuro-cerebral system is a new and rival claimant for this office. If it comes into the field with the disclaimer of any intention to entertain the question of the real conditioning of the phenomena of consciousness, it not only abdicates its claim to be a positive science, but it leaves the field open to the former rival claimants, and that to the great detriment of metaphysic, which is thus left to contend single-handed against those formidable adversaries. Physiological psychologists really ought to have the courage of their opinions.

This leads us to the second of the two failures, the two insufficiencies, noted at the outset, of M. Flournoy's great principles of psychology; I mean their insufficiency to keep psychology clear of conceptions and questions which M. Flournoy himself would designate as metaphysical. At page 118, he quotes a long passage from Professor Wundt's "Essays" (Leipzig, 1885), p. 115-116, which ends with the following sentences (I translate from M. Flournoy's French translation): "Accordingly the naturalist, who, in his own domain, treats the world with justice as a reality independent of our mental phenomena, can never lay down anything beyond a *parallelism* between these latter and the concomitant physiological functions. This parallelism involves the supposition that the phenomena of the psychical life form of themselves a closed totality (*un tout fermé*), a chain of causes and effects as complete as that which exists in the material world; but just because we are thus in presence of two causal series, each complete on its own account, there can be no passage from the one to the other."

To this view M. Flournoy hastens to give his unqualified adhesion in the very next paragraph, which concludes as follows: "To psychical facts we must assign psychical causes and effects; to physical facts physical causes and effects; and never seek in one of these spheres the *cause* or the *effect* of that which takes place in the other (unless we take these words not in the strict but only in the figurative sense, as meaning simply the *concomitant*)" (p. 118-119).

Here then, it is to be supposed, we have M. Flournoy's real meaning; at last the secret is out. Now we see what is meant by his so modestly restricting physiological psychology to a concomitance (excluding dependence) of two sets of heterogeneous phenomena. The fact of dependence (commonly called causality) which is excluded from the relation between neuro-cerebral process and consciousness, is admitted between the phenomena of each sphere taken by themselves. But these spheres or sets of heterogenous phenomena together constitutes the subject-matter of physiological psychology. The fact of dependence, therefore, though so carefully excluded from the relation between the physiological and the psychical, nevertheless comes into the subject-matter of physiological psychology, and that in two places instead of one. And since the psychical states, between which it is admitted, are an essential part of the subject-matter of the science, the whole question concerning the genesis of, and nexus between, psychical states is not only re-opened, but is also re-introduced into physiological psychology, and that with the additional announcement that physiology can contribute absolutely nothing to its solution. In short, physiological psychology, in M. Flournoy's hands, simply and expressly abdicates in favour of what M. Flournoy would call metaphysical hypotheses concerning the nature of the soul. As to his proposed line of demarcation between physiological psychology and metaphysic, it vanishes completely and irrecoverably.

Moreover, the idea of a causal nexus between psychical phenomena taken alone—an idea which, as we have seen, Professor Wundt expresses by calling them "a closed totality, a chain of causes and effects,"—completely breaks down when applied to the facts. To revert to this simple instance of the light of day and the darkness of night, which as already stated are strictly psychical phenomena, it is impossible to suppose that the light of day is caused by the darkness of the preceding night, or the darkness of night by the light of the preceding day. And equally impossible is the supposition that either of them is caused by the psychical events which have taken place in the minds of those who have lived through the day or the night preceding them—that the dreams, say, of sleepers are among the causes of morning. Obviously their cause is partly of a physical, partly of a physiological nature. The "closed totality" theory of psychical events is wholly inapplicable to explain the fact of their occurrence.

This deplorable muddle, for so it must be called, is due solely to M. Flournoy's determined exclusion of the fact of dependence from the idea which he forms of the concomitance or parallelism between neuro-cerebral processes and consciousness. He intends thereby, no doubt, to set physiological psychology free from even the semblance of admitting metaphysical matter. But inasmuch as it is a departure from the simple truth of observed facts, it is sure, sooner or later, to issue in more serious and more manifest error. The most singular thing about it is that the idea of dependence is not strictly, as M. Flournoy seems to imagine, a metaphysical, but a scientific idea. In admitting it into his conception of the constitutive principles of physiological psychology he would not have ventured upon metaphysical ground at all, but would have been keeping strictly within the domain of positive science.

M. Flournoy seems to share the view too commonly taken of metaphysic, namely, that it consists of guesses at the solution of problems which have been abandoned by science as insoluble, and in the form which science, before abandoning has given them. If this were so, it is needless to say that the contempt which M. Flournoy expresses for metaphysic would be fully justified. But in reality nothing can be farther from the truth than this idea. Metaphysic is the analytical examination of the knowledge we have of things, both physical and psychical, in its character of knowledge instead of being (as science is) an examination of things physical and psychical in their character of given objects. It thus deals with the very same phenomena as positive science, but it deals with them on their subjective side, which is but varying the phrase used above, in saying that it deals with our knowledge of things physical and psychal. Now dependence, or what is commonly called causality, belongs only to things as given objects; it is the real order *of their occurrence*, both in co-existence and in sequence, expressed as subject to constant laws; it is therefore a scientific and not a metaphysical idea; to ascertain this order in all departments of the course of nature is the whole business of science. What metaphysic has to do with it is merely to ascertain what our knowledge of it is, what the meaning of the term is, and what evidence we have for its reality in that meaning. The only sense in which it can be said that metaphysic goes beyond, or is the completion of science, is this, that it gives the obverse and subjective side of the same phenomena which science treats on their objective side, as things or objects already given and existent. It in no way attempts to give a more complete scientific answer to scientific problems than science itself can give.

Psychical states and events, which as we have seen, are one half of the subject matter of physiological psychology, are no exception to the universally applicable distinction just laid down. They also, like physical things, can both be treated as given objects, and have the laws of their occurrence ascertained, as they are ascertained by psychology, and also be treated as pieces of knowledge or experience, that is, as they are actually known or experienced in consciousness, consciousness being itself a process of knowing. If, therefore, it is only as given objects, dependent on other given objects, that they come within the scope of science, it follows that the laws of their genesis, evolution and combination, as so depending, are what a scientific psychology has to investigate and ascertain.

But if this be a true account of the relation between metaphysic and positive science, it is obvious that, so long as this relation is kept in view, there can be no clashing between the two pursuits, not even when the phenomena treated of are those of consciousness. Metaphysic and psychology have each its own method, its own purpose, and its own principles, in dealing with them. Metaphysic treats them as part and parcel of our total knowledge of the Universe about us, psychology treats them in respect of their connection with the organisms, to which they belong. Nor does this distinction relate only to those states of consciousness which are our knowledge and evidence of an external and material world; but the states of consciousness which constitute what are called the phenomena and laws of Ethic, Logic, Æsthetic, and Theology, also fall into the province of metaphysic so far as their nature and validity are concerned, and into the province of psychology with respect to their genesis in the neuro-cerebral organism.

The line of demarcation between physiological psychology and metaphysic is drawn clearly and precisely by means of the above distinction. And in consequence of it the two pursuits are placed on the footing of friendly allies, all reason for dispute and hostility being removed. The basis of the alliance is found in the simple fact, by which also the above distinction is justified, namely, that the whole of the *causæ cognoscendi* of all phenomena whatever lies in consciousness—the whole of their *causæ existendi*, so far as we have any means of positively tracing it, lies in matter. If there were no consciousness, matter could not be known to exist; if there were no matter, consciousness could not exist in human organisms. Physiological psychology rightly takes both consciousness and organisms as given existents or realities; but it is false to its own principles if it denies, either that the relation between them is one of dependence, or that this dependence is the dependence of consciousness on the organism. If facts should be observed which compelled us, in theory, either to reverse this order of dependence, or to admit that, though there was simultaneity there was no dependence, then there would be an end of physiological psychology.

SHADWORTH H. HODGSON.

Des Phénomènes de Synopsie (*Audition Colorée*) *; Photismes ; Schèmes visuels ; Personnifications.* Par TH. FLOURNOY, M.D. Paris: F. Alcan, 1893.

In this volume Prof. Flournoy gives us the results of inquiries carried out partly by himself, partly by a student of medicine, M. E. Claparède, into the curious phenomena known as coloured hearing and allied phenomena. These have already been dealt with by Bleuler and Lehmann, F. Galton, Gruber and others. At first the range of phenomena investigated was limited to vivid images of particular colours immediately excited or concomitants of particular sensations of sounds, articulate and musical. This field has gradually been enlarged by the recognition of a similar excitation of visual images or photisms by other than auditory sensations, and also of the excitation of uncoloured visual images, as those of particular schemes and diagrams in connection with the series of digits, days of the week, months, &c. Prof. Flournoy still further extends the area of phenomena by including what he calls personifications, that is, the suggestion of particular types of human face or complete figures, by the numerals I., II., III., &c. To these phenomena collectively, he gives the name of visual synthesis or "synopsie." The setting forth of these various divisions of synoptic phenomena is contained in chapter i.

The next chapter discusses the psychological principles by which it is proposed to account for the phenomena. These are reduced to three: of these the first is affective or emotional association; that is, similarity of emotional tone or analogy of feeling, as when a high musical note excites the visual image of a bright colour. The second is habitual association, or the effect of repeated presentation together (association by contiguity), as when the months and the days are represented in columns resembling those of an almanack or calendar. The third principle is privileged association, by which is meant the effect of specially interesting and impressive juxtaposition, especially in early life. In the nature of the case these associations, dating from a remote period of life, cannot in general be certainly ascertained. The author recognises that it is as yet practically impossible to say how much is referable to the action of one principle, how much to that of another. He tells us that he has been unable to find a subject who can indicate with exactness the origin and cause of his photisms.

Chapter iii. begins the detailed examination of the phenomena by an account of alphabetical photisms, *i.e.*, the visual images suggested by particular vowels and consonant sounds. A large proportion of those who have coloured hearing at all have it in connection with vowel sounds, whereas, only about one-fifth of those who hear the vowels coloured attribute colour to consonantal sounds. Coloured hearing seems to be a very variable capacity, some displaying it more widely with reference to letters, sounds, days, &c., others more narrowly with reference only to particular excitants, as certain letters, certain sounds, &c., and some exclusively with reference to one letter, one month, &c. The complexity of the excitant (inducteur), *i.e.*, the auditory, visual, articulatory and manual (scriptive) elements in letters and words is then discussed. Thus the various degrees of intensity of the indexed visual image are distinguished, as they range from localised hallucinatory quasi-perceptions through images of varying degrees of vividness down to vague non-pictorial thoughts. With regard to the photisms excited by vowel sounds it is shown that amid wide diversities of individual experience there is observable a certain amount of constancy or uniformity. This applies to the particular hist attributed to the vowel, as also to the degrees of brightness of the colour. Thus, while 52 persons see the *a* as white, only 4 see it as gray, while 50 see it as red, only 3 see it as green. With respect to brightness the amount of agreement is much larger. Thus, while 114 persons visualise the vowel *i* as bright, only 18 visualise it as dark. Photisms excited by the diphthongs, the consonants, and by whole words are dealt with in a similar detailed manner.

In chapter iv. the writer gives an account of other photisms, viz., those induced by musical sounds, by odours and tastes, &c. The notes of the diatonic scale appear each to have its proper colours to a considerable number of the subjects examined, but there is great diversity as to what particular colour characterises a given note. Thus the third (mi) appears now as rose, now as blue, and so forth. The colour-photism seems to be affected by the timbre of the tone. As to brightness there is a general agreement that high tones are bright, low tones dark.

These musical photisms appear to be largely due to affective association; yet in certain cases the action of (contiguous) association is traceable as when a tone is given the same colour as that of the instrument producing it, *e.g.*, when the tone of a piano is seen as white and black, that of a violin as the colour of the wood. Notwithstanding the well-known power of odours in exciting vivid recollections, these sensations as well as tastes but very rarely induce photisms. Under the head, "Photisms of sympathy," we have described the curious fact that in the case of at least one subject persons who are antipathetic excite a disagreeable colour (violet), whereas those who are sympathetic excite an agreeable colour (rose or red). Names of persons, places, days of the week, &c., appear also to excite photisms in a certain number of cases. The same is true of the numerals, 1, 2, &c. The chapter concludes with a reference to non-visual synæsthesis, as when the tickling of a certain part of the back of the left hand provokes a disagreeable sensation on the left cheek which produces a grimace and a movement of the hand to the spot. But one notices here the want of a recognition of the large area of such "Mitempfindungen," as described by Lotze and others.

The two chapters on schemes and diagrams add interesting details to the account given by Galton. Very curious is the series of increasing and decreasing ovals or circles corresponding to the succession of the months. In certain cases, as that of the French novelist, Pierre Loti, a month assumes a more concrete or pictorial symbol. The diagrammatic representations of numbers, days of the week and months are very various, but are too complicated to be described here. These geometric arrangements are in many cases definitely externalised and localised, as before the subject, above his head, in a definite portion of the visual field. The writer shows that these diagrams are useful as giving intuitive clearness to the mental representation of time sequences. The origin of the diagrams is thus explained tentatively by means of the principles given above.

Chapter vii. describes personification, that is, the tendency in certain persons to picture numbers, names of the months, &c., under a definite concrete form, as when 1, 2, 3, are said to be children not definitely characterised playing together.

The volume concludes with a chapter on the evolution of "synopsies," in which the writer traces out the variations in the phenomena with age, and discusses the question whether they ought to be classed as pathological. He decides that while abnormal, they cannot be called pathological. A supplementary chapter is added, dealing with the case of M. Grüber, who sent a memoir on the subject to the Congress of Experimental Psychology, held in London in August, 1892, and is engaged in making further statistical inquiries.

This slight sketch of Professor Flournoy's volume may suffice to show that it is an interesting contribution to the study of some of the most curious psychical phenomena lying on the confines of normal and abnormal life. As such it deserves the attention of psychologists and pathologists. What is most needed in the present stage of the inquiry is perhaps a wide statistical examination of children. It seems highly probable that, as in the case of a girl known to the present writer, coloured hearing and similar phenomena are often clearly marked in the early years, and disappear with adolescence. It would also be important to find out to what extent the phenomenon accompanies susceptibility to visual hallucination, to vivid imagination generally, as noted, say, by Galton's questions.

JAMES SULLY.

Critical Digest.

HYSTERIA AND NEURASTHENIA.

BY J. MICHELL CLARKE, M.A., M.D., M.R.C.P.
Physician to the Bristol General Hospital.

I.—Pathogenesis of Hysteria and Neurasthenia.
II.—Symptoms, &c., of Hysteria.
III.—Hysterical and some other Functional Disorders of Movement.
IV.—Hysterical Disorders of the Organs of Vision.
V.—Hypnotism, Hysterical Somnambulism, and Double Consciousness.
VI.—The Association of Hysteria with Organic Disease of the Nervous System.
VII.—The "Traumatic Neuroses."
VIII.—General Features of Neurasthenia, after Löwenfeld.
IX.—Treatment of Hysteria and of Neurasthenia.

"Pathologie und Therapie der Neurasthenie und Hysterie," dargestellt von Dr. L. Löwenfeld, Specialarzt fur Nervenkranheiten in München. 2 vols. J. Bergmann, Wiesbaden, 1894, pp. 744.

"Hypnotisme et double Conscience, origine de leur étude et divers travaux sur les sujets analogues," par le Dr. Azam, Prof. hon. à la faculté de Médecine de Bordeaux, &c., avec des préfaces et des lettres de MM. Paul Bert, Charcot et Ribot. Paris, Felix Alcan, 1893, pp. 375.

"La Névrose Traumatique. Etude Médico-légale sur les blessures produites par les accidents de chemin de fer et les traumatismes analogues," par le Dr. Ch. Vibert, expert près le tribunal de la Seine. Paris, Libraire J. B. Baillière et Fils, 1893.

"État Mental des Hystériques. Les Stigmates Mentaux," par Pierre Janet. Paris, Rueff et Cie., 1893, pp. 233.

References to other pamphlets and papers abstracted will be found at the end of this digest.

I. Papers on the Theory of Hysteria and of Neurasthenia.

I.—Dr. Lowenfeld's work is the most important recent contribution to the literature of hysteria and neurasthenia. It is clearly written, in an interesting style, and the author is thoroughly conversant with the most recent advances in the knowledge of these neuroses. It is, in our opinion, the best work on the subject. It is marked by originality of thought, soundness and independence of judgment. The author has done full justice to the works not only of German writers but of those of other nations, his criticisms are conceived in a broad and liberal spirit, and his own experience enables him to form a just estimate of the observations of other workers in this field. In the following pages a somewhat full account is given of the section on neurasthenia because there is much information in this section which is either not to be obtained elsewhere, or exists only in scattered papers. On the other hand, with regard to the section on hysteria, only those parts of this section which contain novel matter, or deal with points which are still a subject of dispute are here considered. It is not to be inferred from these necessarily fragmentary references that the account given of hysteria in this book is otherwise than full and complete, and indeed, in all respects a most valuable one.

In commencing his book with some general considerations, Dr. Löwenfeld observes that hitherto in spite of numerous efforts, we know of no anatomical changes as the cause of either neurasthenia or hysteria. Clinically it is difficult to separate the essential and distinctive from the non-essential symptoms of neurasthenia, and to isolate a distinct type for it. In hysteria, the numberless attempts to get a satisfactory definition, and to bring its symptoms into a definite form, have failed.

There are two chief views on the place of hysteria amongst nervous diseases—that it is (1)a general neurosis, a disorder of the whole nervous system, or (2) a psychosis, a disorder of the organ of perception, *i.e.*, of the grey matter of the brain. This latter view has been pushed furthest by Möbius, who observes that hysteria comprises all those morbid changes in the body which are brought about through representations (ideas). If this conception is adopted, then hysteria depends on a functional disturbance of the cortex, and further, a psychical therapeusis is the only rational mode of treatment for it.

Such a dependence on idea, or on psychical processes in general for all hysterical symptoms is not proved, nor even shown to be probable; an analogous mode of reasoning would be that, because influenza is often the cause of neuralgia, therefore all neuralgias arise from influenza.

The origin of hysterical symptoms from idea, is, in the author's opinion, only true for a limited number of them, and the hypothesis of the "idiogenesis" of symptoms is not an "open-sesame" to the secrets of all hysterical processes. If the idea of inability to move a limb can cause paralysis, the cerebral process in question consists of an inhibition of the centres concerned in the movement by this idea of powerlessness, so that other stimuli can no longer affect them. But if severe hysterical convulsions with a possibly fatal issue, attacks of profound syncope or fever can be produced through idea, then the cerebral processes connected with such symptoms defy interpretation; in these cases knowledge of the cause fails to explain the results. There must be a pecular condition of the nervous system to render such consequences possible, and it appears to the author quite immaterial whether this condition is produced by idea or any other cause whatsoever. The effects of a gunpowder explosion depend on the quantity and quality of the powder, and not on the composition of the spark which ignites it. The altered condition of the nervous system is a *sine quâ non*, and is of so great importance that the exciting cause of the symptoms sinks into insignificance.

The fact that in hysteria psychical agencies may cause functional disturbances, which in health could not be similarly aroused, is not enough to constitute hysteria a psychosis. The special disorders which occur in hysteria are not related so much to anomalies in the formation of ideas themselves, as to their effects, connected as these are not only with the condition of the grey cortex, but also with abnormal irritability of the subcortical centres. Further psychical disturbances in hysteria, with the exception of delirium are not of such a kind, nor so constant, as to warrant it being considered a psychosis. There is no proof that the development of the hysterical disposition depends entirely, so far as the central nervous system is concerned, on abnormal conditions of the grey matter. The predisposing causes of hysteria are, in great part, of such a nature as to affect the whole nervous system. If hysteria is not a psychosis and is not related only to changes in the cortex, but also to changes in the lower centres, it follows that the author allies himself to the older view that hysteria is a general neurosis, and that its fundamental character is, as is the case in neurasthenia, a state of irritable weakness in which, as a rule, irritability is in excess of weakness.

Nervousness.—Transitional forms connect hysteria and neurasthenia with other neuroses and many pyschoses as well as with conditions which cannot be considered as distinct disease, namely, those which are entitled from the point of view of symptoms as "nervousness" or irritable weakness, and from that of etiology as the neuropathic constitution or disposition. "Nervous" and "nervousness" are used by the public as convenient terms to express a variety of conditions, and, unlike that of hysteria, convey no sense of reproach. The most severe and the lightest affections in the domain of neurology are alike called "nervous." Science cannot make use of these terms in the wide sense that custom has given to them, and it is necessary to inquire what is to be definitely understood and defined by "nervous" and "nervousness." Under certain conditions—in the wear and tear of ordinary life—a sound man may become nervous; in some circumstances not to be nervous would prove the possession of an abnormal or unusual condition of the nervous system. To this transitory and, so to say, physiological nervousness stand opposed the permanent, life-long, frequently congenital states of nervousness. In the latter, increased reaction to external agents in the form of psychical disturbances is of chief importance. Irritations which produce trifling effects in the healthy, give rise to disagreeable and even painful sensations, accompanied by motor, vasomotor and secretory reactions. Emotional processes produce more easily far greater effects on the whole central nervous system than in the healthy.

With increase of sensibility occur the symptoms which point to weakness and excessive irritability of the functionating centres. The facts at command do not allow this increased irritability and lessened power of work to be wholly localised in the same elements or parts of the central nervous system. Lechner explains such symptoms by a disturbance of the normal relation between muscle and nerve-irritability. That the exhaustion of nervous persons can in no way be wholly or even generally imprinted on the central activity is shown by the fact that there is often no diminution of the intellectual powers. The author enumerates the different symptoms by which nervousness is shown in the child and in the adult, and points out that strong, well-built men are affected by it as well as those of more delicate organisation; that under favourable circumstances good, often first-rate work is done in the fields of art, science and industry by such individuals, and that the condition is to be regarded as one midway between health and disease. In favourable surroundings the nervous nearly attain to good health, but under unfavourable ones—prolonged strain and anxiety—a farther development of the condition in the direction of disease occurs, and the symptoms of hysteria or of neurasthenia, that is to say, of pronounced disease, appear. Neurasthenia and hysteria do not always, however, thus develop out of an antecedent nervous condition, they may be directly excited by severe shocks, either physical or mental, such as occur in railway accidents.

Distinction between Hysteria and Neurasthenia.—If we inquire how the distinction between neurasthenia and hysteria originated, seeing the similarity of their basis and fundamental characters, the reply is that it has arisen rather in the mode of growth of our knowledge than in the nature of these affections. The chief characters of hysteria have long been recognised; the symptoms of neurasthenia, until Beard in recent times accurately described them, were confounded with those of very different conditions. Curious distinctions between the two affections have been drawn in recent years, but the physician who is concerned not with the theories of disease, but with the actual sufferers from it, learns by experience that hysterical symptoms often occur in typical cases of neurasthenia and *vice versâ*, and that the distribution of the two neuroses in great part overlaps. There is certainly justification for the use of terms such as neurasthenic-hysteria or hystero-neurasthenia, and many authors attribute no great importance whether in one or the other case the designation neurasthenia or hysteria is used. The conception laid stress on by French writers that neurasthenics with hysterical symptoms and hysterics with neurasthenic symptoms are suffering from two different disorders at the same time seems far fetched. After all, our present knowledge indicates the resemblance of hysteria and neurasthenia in essential points, with varying alterations in the intensity and in the extent of the disturbance of the nervous system.

In numerous neurasthenical and in many hysterical persons, a melancholic or hypochondriacal disposition is met with; when this predominates over the other symptoms, we no longer speak of hysteria or of neurasthenia, but of hypochondriasis. Under the last title are included different conditions—hypochondriacal psychoneurosis or hypochondriasis in the strict sense of the term, hypochondriacal melancholia and monomania. The two last forms of psychical disturbance are distinguished by the intensity and persistency of the melancholic disposition and by the presence of hallucinations. As regards the hypochondriacal psychoneurosis, Gugl has lately insisted that all those cases are to be considered as hypochondriasis in which the mental distress is altogether out of proportion to the actual ailments. The author only recognises the influence of hypochondriasis in so far as the psychical factor—hypochondriacal imaginations and feelings—dominates over the other symptoms.

Theory of Neurasthenia.—Meynert explains the symptoms of neurasthenia as due to the reciprocal influence of irritability and weakness of particular parts of the brain in inducing change in one another. The nutrition and irritability of the cortex and of subcortical areas respectively, may be very different in the same parts of the brain at the same time, and in weakened heart-action the nutrition of the subcortical fails more readily than that of the cortical centres. Long before disturbances occur in the cortex itself, a weakening of the cortical centres is shown by loss of their inhibitory influence on the subcortical, projected into the hemispheres in the form of hallucinations and convulsions. Thus the activity of the subcortical centres, and of subcortical vasomotor centres is increased, gives rise to hyperæmia of the cortex, and hence to irritable weakness of localised parts of it. Meynert further considered the irritability of nerve-cells as a function intimately bound up with their nutrition, and consisting in a molecular attraction on the surrounding plasma. The psychical element in "nervous shock" consists in the loss of this molecular attraction, and these molecular changes are not to be considered as altogether mechanical, but as chemical (nutritive), being liable to take place rapidly on account of the instability of the phosphorus compounds.

Kowalewsky relies on some experiments of Anfimow and Sadowsky, who showed that long-continued electrical irritation of peripheral nerves induces structural changes in central nerve-cells (coagulation-necrosis and vacuolation). Excessive work of nerve elements causes (1) defective nutrition of their protoplasm; (2) auto-intoxication (*a*) from increased katabolic, and (*b*) from insufficient anabolic processes. Accompanying this auto-intoxication there is general insufficiency of tissue-oxygenation, and retrogressive blood-changes which further damage the nerve-elements, and through impoverishment of the blood interfere with general nutrition. Hereditary neurasthenia is attributed to poisoning of the central nervous system with ptomaines and leucomaines from hereditarily defective processes of metabolism.

Lechner holds that neurasthenia results from disturbance of the normal relationship in action between muscle and nerve. Dr. Löwenfeld observes that neurasthenia would be better understood if the changes which take place during the stage antecedent to physiological fatigue were better known. The changes in the nervous system in neurasthenia must be molecular, and are never gross. During the waking state full restoration of used up material and removal of the products of the activity of central nervous system does not occur. If the nervous capital is to remain the same, it must be restored during sleep. In neurasthenia the accumulation of waste products is so great that the period of sleep is no longer sufficient to fully remove them, so that a chronic over-lading of the nervous system takes place. In this way a neurasthenic condition is produced, and Kowalewsky's theory holds good for a great many cases, but the occurrence of neurasthenia from physical or mental shocks, from insolation and poisoning cannot be thus explained. If, however, a severe commotion of the nervous elements can reduce their metabolic activity, an accumulation of toxic products may be supposed to take place.

These disturbances of chemical tissue change in neurasthenia affect different parts of the central nervous system in different persons, but all parts are so bound together that one centre cannot be disturbed without affecting another. Vasomotor disturbances, started by slight and normally inadequate causes play an important part in the symptomatology of the disease. Lastly, the signs of increased irritability of nerve centres are not merely due to diminished inhibition from higher parts, but to an original abnormal increase of sensibility to external influences.

Dr. Joseph Breuer and Dr. Sigm. Freud[1] have been struck with the importance in cases of hysteria of ascertaining the causes and the phenomena immediately preceding the onset of the hysterical symptoms. Partly from the patient's forgetfulness or reluctance to recall a disagreeable experience the most thorough examination generally fails to elucidate these points, and it is necessary to arouse in the patient's mind by means of hypnosis the memory of the symptoms experienced at the onset. Then the connection between the latter and the symptoms present at the time of examination comes out in the clearest and most convincing manner. Observations made in this way are worthy of record, both from the theoretical and practical points of view.

In traumatic hysteria the connection between the exciting cause and the resulting symptoms is evident, and it is clear also in cases of hysterical attacks in which the same prodromal hallucinations, derived from the patient's surroundings, appear in each attack. The authors' experience is that an equally close connection with the exciting cause exists in the various symptoms which pass for spontaneous or idiopathic manifestations of hysteria, and that all varieties of hysterical phenomena (*e.g.*, neuralgias, epileptiform fits, paralyses, contractures, anæsthesias and special sense affections) can be referred to similar ætiological factors. The disproportion between the cause—acting for once only—and the resulting permanent symptoms is quite as marked as in traumatic hysteria; often the disturbing event has happened in childhood. Sometimes the connection between symptoms and cause is as clear and direct as possible, in other cases it is not so definite, but consists in a symbolic relation, so to speak, between the cause and the pathological phenomena, such as the occurrence of neuralgia or disgust for food as the consequences of moral pain and loss of appetite respectively. In other symptoms, again, *e.g.*, hemianæsthesia and contraction of fields, no such connection can be traced.

Such observations appear to show, as regards the pathogeny of the cases, the analogy of ordinary hysteria to the traumatic neurosis, and to justify an extension to the former of the current conception of traumatic hysteria. Psychical shocks (traumata) act as the causes of many, if not of most hysterical symptoms. Any experience which entails emotions of shame, terror, dread, &c., may be such a cause, its potency depending entirely on the sensibility of the person affected. Further, in ordinary hysteria, in the absence of more powerful agencies, a series of minor shocks may by summation be a potent cause. Again, conditions, which acting alone would be powerless, may produce hysterical symptoms if they happen to act in conjunction under favourable circumstances.

The causal connection of the physical shock with the resulting hysterical phenomena is not merely in the French sense one of "*agent provocateur*," after whose action the phenomena are capable of further development. The shock and the remembrance of the shock is rather to be considered as a "foreign body," which can still be potent a long time after its occurrence as an exciting agent. Proof of this is found in the fact that if the remembrance of the original cause or causes of the symptoms can be clearly aroused, the emotion which at the time of onset accompanied them is also called up in the patient's mind and that if the patient then pictures to himself as fully as possible what took place, and describes his sufferings aloud, the hysterical symptoms described disappear and do not return. All varieties of hysterical symptoms may be treated in this way. The authors consider that the efficacy of this method of treatment lies in the patient's giving expression to his sufferings, and not in a suggestion arising from his expectation of being cured by the procedure. They conclude that the exciting causes of the events immediately prodromal to the onset of hysterical symptoms, when recalled to the memory after long intervals, can act powerfully and directly in relieving those symptoms, just as under ordinary circumstances a painful occurrence in the past life, when called to remembrance may excite a flow of tears.

II. It is strange that long past events should have so great an effect that the memory of them should not have faded away. In this respect the occurrence or not at the time of onset of an energetic reaction against the disturbing force is of chief importance. By the term reaction is understood the whole group of voluntary and involuntary reflex actions—from tears to acts of vengeance—by which in common experience the emotions discharge themselves. The emotional after-consequences are attenuated in direct proportion to the thoroughness of this reaction. If an injury has to be borne in silence and any show of resentment suppressed, the psychical and physical consequences are more intense. Full discharge of an emotion may be accomplished by deeds or by their substitutes—words; for instance, relief from the burden of a painful secret is obtained by confession or confidential communication. When such outlets are prevented the memory of a painful occurrence is intensified and bound up with distressing emotion. This reaction (Abreagirung) is not the only kind of relief at the disposition of a sound psychical mechanism which has sustained injury. The painful remembrance may enter into complex associations, be corrected by later representations, or annulled by being stored up with memories of experiences of a contrary kind. A remembered mortification suffers correction by the re-adjustment of subsequent events, by sense of personal merit (self-esteem), and in healthy minds disappears through such associations. Painful experiences with their emotional accompaniments may remain fresh and active for an extraordinary time. These impressions are either completely absent from the thoughts of the patient in his normal mental state, or are present in only the most rudimentary form; they correspond to injuries insufficiently "reacted" against, and may be divided into two groups: (1) in which, from the nature of the loss or injury, reaction is impossible (*e.g.*, death of a beloved relative, social hindrances); (2) in which reaction is impossible on account of the patient's mental state at the time (*e.g.*, paralysing influence of terror, partial hypnosis). Both kinds may and frequently do occur together. These pathogenic re-representations remain fresh, with unimpaired power of affecting the emotions, because they do not undergo the ordinary wearing away of such emotional disturbances through "reaction" and association.

III. Hysterical phenomena being thus regarded as developing out of psychical (moral) shocks the abnormal state of consciousness in which such pathogenic re-representations arise must next be considered, and the fact emphasized that the memory of the essential causal factor is only to be discovered during hypnosis. That division of consciousness which in certain classical cases has been described as "double consciousness," exists in rudimentary form in every hysterical person, and the tendency to this dissociation and to the development of an abnormal state of consciousness is the fundamental phenomenon of the neurosis, and may be briefly entitled the "hypnoid state." These hypnoid conditions correspond with hypnosis in one respect amongst many points of differences, namely, that representations which arise during them are very vivid, but cut off from associative connection with the other contents of consciousness; they are, however, associated with one another, and vary in kind and degree just as hypnosis may vary from somnolence to somnambulism.

Such hypnoid states form the soil in which morbid recollections with their physical consequences grow. The two together make up the hysterical disposition, but a severe injury (traumatic neurosis), painful suppression of feeling (sexual affections), can in healthy persons cause a separation of groups of perceptions and thus produce a psychically-acquired hysteria. These hypnoid conditions may develop out of the day-dreams so common even in healthy people, and for which the feminine occupations give so much opportunity. Like hypnotic suggestions, they are able to affect the bodily health more powerfully than ordinary perceptions.

Similar considerations apply to hysterical attacks. In the stage of delirium in the fully developed attack, there is an hallucinatory reproduction of the image of memory of the chief injury or group of minor injuries. In fits which consist of motor disturbances only, if one succeeds in putting oneself *en rapport* with the patient whilst in a cataleptic condition, or attack of sleep, or still better if the convulsion can be evoked during hypnosis, then the remembrance of the injury or group of small injuries which lie at the bottom of the attack can be discovered and made clear. These images of memory correspond to those which have been given as the basis of the permanent symptoms of hysteria. Our observations have proved further that such remembrances which have previously provoked fits, become powerless thereto, if during hypnosis a "reaction" against them and an "associative correction" of them can be brought about.

It follows from the foregoing, that in hysteria the groups of perceptions which arise in the hypnoid states are connected with each other, but shut off from other associations, and thus form a rudimentary second consciousness. The permanent symptoms correspond to a disturbance of innervation of the organs formerly under the control of the normal consciousness by the intrusion of this second state, and the hysterical attack, to a still higher organisation of the latter, by which it gains a complete mastery over the whole personality. Charcot has enunciated the doctrine that an hysterical attack forms the rudiment of a secondary state of consciousness. The normal consciousness is not always completely abolished during a fit; it can perceive the motor phenomena of the fit, whilst the psychical occurrences are beyond its ken.

Frequently there is a kind of equilibrium between the different psychical states present in the same person. Fit and normal life alternate with each other without influencing one another. The attack may appear spontaneously, as is the case with normal images of memory, or may be provoked by irritation of hysterogenic zones, or by the similarity of a new experience, to the pathogenic one, just as a particular image of memory can be aroused through the help of its associations. The authors' mode of treatment is now intelligible. It removes the efficacy of the original disturbance which has not been "reacted" upon, by affording through speech an outlet to the pent-up emotion, and by bringing it into "associative correction." The authors think that this method is productive of great therapeutic advantage; they do not profess to cure the disposition to hysteria, but to prevent the return of hypnoid states. In the "stage of production" in acute hysteria it is valueless, but is effective in removing fits or other persistent symptoms.

Prof. Janet[2] remarks that formerly every writer on hysteria had to give an account of a number of definitions of the disease; latterly the tendency has been, from the infinite variety of hysteria, to shirk a definition of the disease, or to declare it impossible to define. Thus the authors of recent important books on the neurosis, *e.g.*, MM. Babinski, Pitres, and G. de la Tourette, attempt no general definition, but content themselves with enumerating certain characters by which hysterical phenomena can be recognised as such. But although such a method has its advantages, inasmuch as it allows the phenomena in isolated cases to be studied thoroughly without distraction by theories, yet, on the other hand, it has the inconvenience of producing a very precise idea of an isolated character and a very vague one of the disease itself, and prevents agreement between authors who may believe themselves to be writing of like phenomena, and yet on account of the different teaching of various schools, may be in reality describing distinct ones.

No one expects a definition to explain the essence or very nature of the thing defined; it is sufficient if it expresses in one phrase the majority of the known facts. Like all scientific theories such a definition must be provisional since knowledge of facts constantly increases, but it will hold good until replaced by a simpler one embracing not only the old but the new facts.

1. *Hysteria a Disease of Representation.*—Such a definition should put first the dominant character, and group the other facts around it. The ancient definitions selected as essential a physical character as the name hysteria sufficiently indicates; then Brachet[3] made the convulsive attack the predominant symptom. This was better and more comprehensive, but took no account of the inter-paroxystic period. Briquet's book is the meeting place of the physical and moral conceptions of hysteria; according to him, hysteria is a disease of the emotions, and here is the starting point of the psychological theories of hysteria. Charcot first enunciated these precisely, clearing the ground by demonstrating the characters which distinguish hysterical from organic paralysis. He then showed from the origin, characters, course, and treatment of hysteria that it is a moral (psychical) malady, and confirmed this view by making use of hypnotic suggestion. "Under certain circumstances paralysis can be caused by idea. By reason of the clouding over of the *ego* produced in one case by hypnotism, in another by nervous shock, an idea once installed, fixed in the mind and ruling there without control, will further develop and acquire sufficient force for objective realisation." Here we have the conception of the influence of suggestion and of "fixed ideas." M. Georges Guinon further elaborated the idea of suggestion, and Dr. Möbius generalised it. He writes: "One

view tends to become established, namely, that hysteria is a psychosis, and its characteristic is a morbid mental state." This mental state manifests itself by bodily symptoms. Hysteria differs, however, from insanity, in that in the latter the actions of the insane person are only indirectly in relation with his delusions, the insane person bestirs himself in obedience to an imaginary order, but in hysteria the relation is more direct, thought is transformed into movement without the mediation of the subject's will. Strümpell, expressing analogous ideas, writes: "Certain over potent representations, certain associations of ideas too easily become the point of departure for a long series of apparently physical accidents." He explains these neuropathic phenomena as dependent on idea (Vorstellungen).

The new definition is then that *hysteria is an aggregation of disorders depending on representation.*

2. *Double Consciousness.* — A great number of hysterical phenomena do depend on fixed ideas, upon suggestion; but they cannot all be thus accounted for. (1) There are two kinds of hysterical tics or spasms: (*a*) those which take place when the patient thinks of them, and disappear during sleep, or when his attention is diverted; (*b*) those which persist in spite of all distractions, and sometimes in spite of sleep. These latter must long ago have passed out of the domain of consciousness. (2) Convulsive attacks, which recur with monotonous regularity, seem to depend on physical causes rather than on any thought or idea of the patient. (3) The more direct mental phenomena, which belong to hysteria, such as delirium and somnambulism, are connected with a fixed idea. In short, putting aside the "stigmata," it is impossible to regard the paroxysmal phenomena (accidents) as due to physical changes produced by representations in consciousness. To change the point of view, we will take "the somnambulistic state" as the main point of the definition. This state in every variety of it has been shown to occur in hysteria; lasting sometimes for weeks or months, and giving rise to "double personality." The only constant and essential character of somnambulism is the forgetfulness of all that has passed in the somnambulistic, when the patient has returned to the normal state. Generally the lost remembrances are regained when the subject passes again into somnambulism, and he may thus live two lives, or have two psychological existences alternately succeeding each other. Somnambulism, from the simplest to the most fully developed form, is always the result and the manifestation of a double personality. Somnambulism can be provoked by various

causes, which, however, do not create or form the double personality, but evoke it when it is already existent. Using somnambulism in this sense, many hysterical phenomena, *e.g.*, periods of double existence or automatic action, states of ecstasy, of catalepsy, of delirium, may be looked upon as specially modified forms of it. Further, M. Charcot has shown that close relations exist between the somnambulistic state and hysterical convulsive attacks; symptoms present in the one are also present in the other; they are both amenable to the same influences, the subjects pass readily from the one state to the other, and memory of them is also reciprocal; the patient remembers in the convulsive attack the features of the somnambulistic state and *vice versâ*. Most often the convulsion is excited by an emotion, dream, or fixed idea which is a part of the second (somnambulistic) existence. A striking feature about hysterical convulsions is that they always take precisely the same form in the same patient; for one individual the events are always the same even during a period of years. Similarly for the somnambulistic state, the sufferer always performs exactly the same actions, with the same automatic regularity. The second existence is often a rudimentary psychologic existence, in which a few sensations and ideas exist, not controlled nor modified the one by the other. The fixed ideas which play so large a part in the waking state in hysteria are still more powerful and develop themselves with greater regularity in the second existence. Examining more closely the features of the convulsive attacks, some of them are obviously reproductions of an event in the patient's life; others, such as simple convulsions, cries, &c., are to be regarded as expressions of the emotions. By throwing the patient into the somnambulistic state, and profiting by the memory he has of the phenomena of the attack whilst he is in this state and only whilst he is in it, it is found that some emotion, as terror, anger, despair, always initiates the attacks. All hysterical attacks are of the same nature, and consist in a more or less complete reproduction in a second existence of a past idea, adventure, or emotion, that is, of a more or less rudimentary somnambulistic state.

The above considerations apply to an important category of hysterical troubles, namely, the paroxysmal manifestations, but fail to account for the permanent symptoms. The author has tried to unite both in one explanation by the study of the manifestations of the second personality in the intervals between the somnambulistic states and between the convulsions. The study of the influence of suggestion in producing post-hypnotic actions first suggested this explanation. The subject has been told to do something in eight days' time, and without any memory of the suggestion, without calculation or reflection, he does it at the appointed time. Instances of the same kind are the spontaneous unconscious actions and automatic writing of mediums. There is thus a second mind existing behind or below the normal one. The facts may be summed up thus :—" These apparently sub-conscious acts are not distinct from somnambulism, but make up a somnambulistic state itself, no longer isolated, alternating with wakefulness, but prolonged without interruption through the waking state. A double personality results, because all psychical phenomena are no longer united in one and the same personal consciousness, but in part remain independent under the form of sensation or elementary images, and are more or less completely aggregated to a new personality, independent of the normal (original) one."

This explains the patient's frequent ignorance of the idea which has provoked the hysterical accident. It is present in the second personality, and its existence can be demonstrated during hypnosis, or as the author has often done by means of automatic writing, but in his ordinary state the patient will be unconscious of it.

Thus hysteria is in a sense a disease of *dissociation* of ideas, and this dissociation plays in it a *role* as important as that of association in normal psychology. " The essential character of this 'disease of disaggregation' is the formation in the mind of two groups of phenomena, the one constituting the ordinary personality, the other, capable of further sub-division, forms an abnormal one, different from the first, and ignored by it." M. Janet observes on this new conception of hysteria that the incomplete state of the normal personality constitutes the hysterical vice and permits the disorderly action of the second personality, *i.e.*, hysterical accidents. The second personality hidden under the first, stronger by so much as the latter is enfeebled, replaces it at the first opportunity and becomes apparent. Breuer and Freud, in the preceding paper, remark that " This division of consciousness that has been clearly demonstrated in some celebrated cases of double consciousness exists in a rudimentary form in every hysterical person, and the disposition to this dissociation, and at the same time to the formation of abnormal states of consciousness—hypnoid states—constitutes the fundamental phenomenon of the neurosis."

3.—*Restriction of the Sphere of Consciousness.*—The above considerations have a general application to the majority of hysterical accidents, but they leave the permanent stigmata of the neurosis almost entirely unaccounted for. These are an essential part of it, and consist mostly of troubles of sensation, memory, and volition, shown in modifications of movement and of character. Some authors have applied the same explanation to these permanent symptoms as to the paroxysmal ones, and accounted for them on the hypothesis of a fixed idea. For the following reasons the author cannot accept this explanation :—(1) There is a precise event at the onset of a paroxysm due to a fixed idea, but there is nothing in the history of the patients to suggest to them left hemianæsthesia or contraction of the visual field. (2) The former are recognised by the patient, whilst they are often ignorant of the presence of the permanent stigmata. (3) Neither in the somnambulistic state, nor by any method of procedure can the existence of a fixed idea relating to the stigmata be shown. (4) The stigmata have characters of which the patient has no conception, whilst the conception of the accident determines the nature of the symptoms. (5) Fixed ideas are of the most variable character, the permanent hysterical stigmata are constant, and have been the same since the middle ages. (6) The tendency to the formation of fixed ideas, the amenability to suggestion, only takes place late in the course of the disease.

These stigmata take the form of enfeeblement or suppression of sensation, memory, or movements. In the case of anæsthesias the sensory loss is not a localised one, but rather a particular manifestation of an enfeeblement of all the functions of the cerebral cortex. M. Janet considers from various reasons that the stigmata are not due to local lesions of sensory terminations, muscles, nerves, or centres. A state of irritable weakness has been admitted by most authors, but a more precise term is desirable. This cerebral weakness is found chiefly in a lack of the power of attention. Want of volition, aprosexia, hesitation and doubt are the essential psychical characters of hysteria. The weakness of attention is so great as not only to prevent intellectual work, but to modify also the normal life and ordinary thought. An hysterical woman, for instance, is incapable of perceiving several sensations at one time. Her attention is easily distracted, as we say. The "field of consciousness" in hysteria is very small, on account of this readiness to distraction, and may often be fully occupied by a single sensation, understanding by "field of consciousness" the largest number of simple or relatively simple phenomena which can be perceived at any one time. This restriction of the field of consciousness is only one manifestation of the feebleness of cerebration, which may be described as a "special moral feebleness," consisting in the impotence of the subject affected to bring together and condense his psychical phenomena, and to assimilate them to his personality. In this way are explained the traits of character present in hysterical persons, their instability, and excitability, their sudden impulses, passing enthusiasms and whims, their depths of joy and despair, and also such permanent stigmata as the anæsthesia. Professor Janet sums up as follows: "That apparently the elementary pyschical phenomena, are as real and as numerous as in normal persons, but cannot on account of the restriction of the field of consciousness, on account of feebleness of the synthetic faculty, be brought together into one single perception."

The doubling of the personality, brought forward above, does not stand in contradiction to, but is rather the immediate consequence of this feebleness of psychologic synthesis. The facts of somnambulism and of sub-conscious states are to be regarded as secondary groupings of neglected pyschologic phenomena; "as if the system of pyschical phenomena which constitutes personal perception in everybody, was in these persons split up, and gave rise to two or more simultaneous or succeeding groups, for the most part incomplete and separating the one from the other sensations, images and the consequent movements which should normally be brought together into one and the same consciousness." Several authors have accepted this view, and M. Pick, for instance, considers "the diminution of the power of attention, the restriction of (personal) perception, and of motive impulse, as the characteristics of hysteria."

4. *Hysteria a Mental Disorder.*—The tendency of modern writers is to regard hysteria as a mental disease (*e.g., Donkin, Art.*, "*Hysteria*," *Charcot and Marie, Art.*, "*Hystero-Epilepsy*" *in* "*Dict. of Psychological Medicine*"). M. Janet fully agrees with this view, but points out that a definition must (*a*) include all the symptoms of hysteria; (*b*) characterise hysteria only and distinguish it from all other diseases. (*a*) Now the psychical aspect of certain symptoms is not at first sight obvious; there are (1) the *visceral accidents* of the neurosis; (2) the vasomotor and secretory troubles; and (3) trophic disorders. Under (1) come palpitations, coughs, hiccoughs, respiratory spasms, numerous digestive troubles, and ischuria; under (2) hæmorrhages vicarious of men-

struation, ischæmia and other vasomotor affections of the skin, with anidrosis or hyperidrosis; and under (3) muscular atrophy, slow growth of the nails in an anæsthetic limb, &c. Most of these apparently physical disorders are, however, amenable to suggestion and to various psychic influences. The persistent emotions of the hysterical are a common ground for physiological and psychical phenomena, and a more perfect knowledge of the psychology of the emotions than we have at present may give another aspect to phenomena now apparently purely physical. Other symptoms must remain unexplained, but if hysteria is a mental disorder the mind cannot be affected without other organs being disturbed, and to these disturbances, secondary and subsidiary to the mental trouble, we must attribute many of the symptoms.

(*b*) Is this conception of hysteria sufficiently precise to distinguish the disorder from other mental disorders? In answering this question the limits of hysteria must be defined. M. Janet is disposed to include under hysteria somnambulism—he speaks of true somnambulism and not of hypnotism; for it is found only in hysterical persons, its symptoms correspond to those of hysteria, and it presents the phenomena of the double personality so characteristic, according to him, of the mental state of the latter. He would also include certain mental states, the results of intoxication from alcohol or hashish, and this leads him to add to his definition of hysteria a certain duration or persistency of double consciousness. Further, he points out the resemblances to hysteria of the symptoms in patients on the confines of lunacy, affections grouped together in France under the name of "délire des dégénérés," in Germany, of "neurasthenia délirante." He thinks a number of these patients belong both to hysteria and to this form of mental unsoundness; there is one common striking character, that is, defect of volition. The mode of dissolution of the mind is different in the "délire des dégénérés" the development of another personality is felt by the patient who speaks of it as "possession"; in hysteria, the new element of a double personality is ignored.

To sum up: "Hysteria is a mental disorder belonging to the great group of degenerations, its physical symptoms are rather vague and consist principally in a general lowering of nutrition; it is especially characterised by mental symptoms, of which the principal is enfeeblement of the faculty of psychic combination (synthesis), a restriction of the field of consciousness; a certain number of elementary phenomena, sensations and images cease to be perceived, they constitute the stigmata; there results a tendency to complete and permanent division of the ego (*personalité*), to the formation of several groups of mutually independent phenomena; these systems of psychical events either alternate or co-exist, giving rise to the convulsive attacks, somnambulistic states, and sub-conscious acts; finally, this defect of synthesis favours the formation of certain parasitic ideas which, under cover of the control of the personal consciousness are developed independently, and show their presence by disorders of the most variable kind, apparently physical in nature, *i.e.*, the accidents or paroxysmal symptoms."

To put the above in two words, "Hysteria is a form of mental dissolution, characterised by tendency to permanent and complete doubling of the personality."

Professor Janet[4] in lectures at the Saltpêtrière makes a further contribution to the psychological side of hysteria; he concludes from his observations on patients that hysterical anæsthesia is not an organic disease, but a mental, a psychical disorder. It exists neither in the limbs nor in the spinal cord, but in the mind represented—if you will—in the cerebral cortex. In the mind one special phenomenon is concerned; hysterical anæsthesia does not consist of an alteration of the elementary sensations which remain unaffected and retain all their properties, but concerns the special operation of personal perception in connecting new sensations one with another and bringing them into relation with the ego; it is due to a failure of this synthesis of psychical elements that the author has elsewhere named (see last paper), psychical or mental disaggregation. Hysterical anæsthesia is thus a disease of the personality (ego).

After having first demonstrated the frequency of amnesia (loss of memory) in hysteria, Prof. Janet points out the analogies between this loss of memory and hysterical anæsthesia. Just as in the case of anæsthesia so in hysterical loss of memory there is no destruction of the elementary mental phenomena, sensations and images; there is merely an impotence, an insufficiency of the central power (highest centres). There is always a perception by the ego, but one incapable of welding the elementary phenomena into due relation with the personality as a whole. At one time presentations (images) are neglected at hazard, and vague continuous losses of memory are produced; at another certain presentations having definite characters seem to pass unperceived, and the most curious "localised" losses of memory occur. He believes that these localised losses of memory are determined by the anæsthesia present at the time, or, rather, by the variations in the affection of consciousness by sensations Very often more complex influences are at work, and this explanation only applies to particular cases. In the third lecture the writer deals with the abnormal susceptibility of hysterical persons to suggestion, that is, to suggestion in its pathological form as a symptom of psychical disorder. He has tried to show that this symptom does not only depend on the laws of association, such as are applicable to all men, but on a peculiar infirmity of the will. This want of will power is present in several mental diseases, and in particular forms the essential symptom of hysteria; it does not consist in the abolition of all voluntary acts in themselves, but is of the same nature as hysterical amnesia and anæsthesia.

Both Dr. P. Blocq[5] and Prof. Grasset[6] deal with Prof. P. Janet's writings on the pyschological origin of hysteria. Dr. Blocq accepts Prof. Janet's theories, and applies them to the explanation of the various phenomena of hysteria. Amongst the permanent "stigmata" of the disorder, he classes on the intellectual side, amnesia and defect of will power. To the so-called condition of "suggestibility" he attaches important consequences. Normally, when an idea is called up, it is utilised in a particular manner, and of its diverse connections only one is voluntarily selected, and the other associations are neglected. The weakening of voluntary control in the hysterical interferes with this process of selection, and a succession of associated images arise, which result in hallucinations and in various actions.

Prof. Grasset, after showing how far Prof. Janet's views elucidate hysterical phenomena, criticises them, and especially his concluding definitions of the neurosis as follows. He says that Prof. Janet's analysis of hysterical psychical phenomena, ingenious and true as it is in the main, demonstrates that this neurosis is a malady of cerebral or more exactly of cortico-cerebral origin. The dissociation of the sub-conscious from the conscious is a pathological action of the cerebral cortex. The conclusion is that hysteria is a neurosis *principally* of cortica origin. Prof. Grasset says principally because he does not regard it as proved that the above-described psychical characters are absolutely constant and universal, but believes that hysteria is a neurosis of the entire nervous system, in which the brain bears the chief part. Hysteria is only a mental affection, if every disease of which cerebral psychic phenomena form a great part is mental, but not otherwise. The hysteric knows that his aberrations are pathological, but the really insane person believes his diseased state normal, and considers his delusions realities. The centres below the level of consciousness may be pathologically dissociated from those concerned in consciousness, without consciousness itself being changed. A further degree of disturbance concerns the latter as well.

Hysteria can only be properly considered a disease of the personality if both kinds of centres, those concerned in, and those below the level of consciousness, are included under this term; if the two are separated it cannot be so described. It is better therefore to continue to use the term mental in the old and generally accepted sense, and to say that recent researches have demonstrated the psychologic origin or cerebro-cortical formula of hysteria, and not that they have shown it to be a mental disease.

Dr. Blocq,[7] in another paper, considers the theory of Prof. Janet that hysterical anæsthesia is a dissociation of re-presentations too exclusively subjective. He explains in the following way (see also a paper by Onanoff on "Unconscious Perception"[8]) the mechanism of perception of sensations and those modifications of it found in hysterical anæsthesia. The afferent impulses which leave sensory surfaces pass to lower reflex centres, and after a first elaboration there to higher centres, where they are brought into complex associations, and a process of selection then takes place which forms the stage of perception. This final synthesis or selective perception ends in the setting in action of a motor centre, movement occurs and the perception is called conscious (apperception) or unconscious according as the sensory impressions consecutive to the movement return or not to the associated centres and are there identified. In hysterical anæsthesia this series is incomplete. The afferent impulse from without passes unperceived because the action of the associated centres is disturbed; some of them enter into fixed combinations with other centres, which fresh afferent impulses are unable to disturb, and thus the process which ends in identification and in conscious action does not take place. This fixed morbid associated action of nerve-centres, which prevents the proper reception of sensory stimuli, may, however, be broken up by a weak psychical agency, such as suggestion, and hence the instability of hysterical anæsthesia. One psychical characteristic of hysterical anæsthesia, which is common to all varieties, and not capable of simulation, is a shortening of the reaction time; thus, in hysterical hemianæsthesia the reaction time is always shorter on the anæsthetic side. This is a valuable objective sign.

Mental State and Character in Hysteria. — Dr. Lowenfeld observes that the traits of character which have been ascribed to hysteria by most writers until recently, are the exception and not the rule; when they exist they are due to the coincidence with hysteria of an inherited mental taint (degeneration). When the mental characteristics of hysteria are spoken of, it should be remembered that six times as many women as men are affected with hysteria, and that the distinguishing features of the female mind as compared with the male consist of a weaker will, and greater predominance of the emotional over the purely intellectual faculties. Hysterical persons often fulfil their duties well as daughters, wives, and mothers, and are not always seeking to attract attention to themselves and their complaints.

He goes on to say that Blocq and G. de la Tourette attribute the variations in the psychical condition of hysterical patients to an increased "suggestibility," and regard this as characteristic of the hysterical mental state. But this is not present in every case, and is not or only in small part an independent primary symptom. In cases of "nervous shock" (traumatic hysteria), indeed, such a state of increased suggestibility (readiness to react to suggestion) similar to that produced by hypnosis does occur. It is conceivable that when such a condition is present it may soon disappear, whilst its consequences, in the form of paralysis or arthralgia may persist. No further hysterical symptoms arise, and this limitation of symptoms, in a number of cases, has led to the conception of a "local" hysteria. Breuer and Freud describe as the basis of hysterical phenomena the occurrence of hypnoid states. If the enquiry be not restricted to a state of suggestibility, but the psychical condition considered as a whole, it is obvious that hysteria has a basis to work upon which varies with the social position, education, favourable or unfavourable circumstances of the patients. Their surroundings must have an important influence on the psychical manifestations, and one must not attribute to the neurosis, symptoms which have in reality an independent origin. The character, nature, and frequency of the hysterical phenomena have a great influence on the accompanying mental state.

Memory.—Many disturbances of memory occur in hysteria which we do not find in neurasthenia, at least in the same perfection. The power of reproduction in memory of past experiences or of acquired knowledge may be impaired in neurasthenia, but the defect concerns all remembrances in the same proportion, and complete gaps for certain groups of events, or things learnt are not found. On the other hand in hysteria such gaps in the memory are not uncommon, and affect generally short, but occasionally long periods, weeks or months of the past life. Thus one patient, paraplegic after an hysterical fit, lost all memory of the preceding three months, and another, who spoke English fluently, forgot it completely as her hysterical symptoms increased. Many patients fill in the gaps in their memory with fabrications, and thus their statements vary as to trivial occurrences, or if they keep to the same story it does not correspond with the actual facts. In the majority of cases in which hysterical patients make false or exaggerated statements, the defective memory must be borne in mind, as a tendency to lie is not a symptom of the neurosis.

Hysterical suggestibility presupposes an increased "auto-suggestibility" (increased readiness to react to intrinsic stimuli). After mental shocks, for instance, an abnormal state of consciousness occurs in which there is increased reaction to suggestions, and this favours the production by auto-suggestion of various physical troubles. This occurs frequently in children who are liable to hysteria, in an epidemic form. The author agrees with Gilles de la Tourette in attaching importance to dreams as a source of auto-suggestion. We may use this increased suggestibility as a means of cure in various ways.

Emotions. Suicide.—Increase of emotional excitability varies much in individual cases, shows no correspondence with the severity of other hysterical symptoms present, and is probably due to the patient's original character and training. With this increased emotional excitablity is connected the tendency to suicide, a real danger in hysteria, and one not to be under estimated. During somnambulistic attacks attempts at suicide may be made, and the tendency to it is especially obstinate in the complication of hysteria with melancholia. Lastly, the suicidal impulse may be of uncontrollable character, and independent of melancholic ideas. Another condition to be mentioned is the restriction of the sphere of interest or of feeling, perhaps associated with intense absorption in an unworthy object.

Dr. Möbius[9] quotes Oppenheim as follows: "The fundamental phenomenon of hysteria is irritable weakness, abnormal irritability and readiness to fatigue. Extrinsic and intrinsic stimuli have a greater effect than in the healthy. The morbific factor is chiefly the greater readiness with which the emotions can act on motor, vasomotor, sensory, and secretory mechanisms. Expressed physically, the molecular movements take place with less internal friction. Sensory impulses cause more powerful stimulation and more easy discharge of centres. Hysterical paralysis exhaustion arising from the relatively over-powerful stimuli; it is a true paralysis resulting from resistance to conduction at the point where the will grasps the motor centres, offering more or less considerable resistance to voluntary movement. Although the individual cannot carry out the desired movement, this can still be accomplished reflexly, or as a part of an associated action. A similar condition to that of hysterical paralysis is present in the healthy when the stimulus to action is not only relatively, but absolutely excessive to the "Labilität" of the nervous system (*e.g.*, "traumatic neurosis").

So far Dr. Möbius finds no essential difference between his views and those of Oppenheim. He thinks that the physical changes in hysteria are brought about by idea. Even in health lively sensations of pleasure or pain may cause physical disturbance (organic change), and in hysteria similar changes may be produced with unusual ease, whilst besides these others may occur which are not generally found in the healthy. The nature of the idea in the above conditions is not important as regards the nature of the disturbance, but in ideas artificially excited (suggestion), the character of the subsequent results can be foretold. Analogous states to suggestion occur in ordinary life, and whether dependent on emotion or suggestion there is a connection of cause and effect between idea and physical changes.

Oppenheim, however, in opposition to the above theory, which has, at any rate, the merit of clearness, goes on to argue that hysterical symptoms arise (1) through emotions; (2) reflexly. With regard to (1) his views tally with Dr. Möbius's, for an emotion is nothing more than an idea associated with lively feelings of pain or pleasure, but he errs in failing to recognise suggestion as a cause of hysterical symptoms. Very many hysterical symptoms, and the recognition of this fact is due to Charcot, are, however, unintelligible unless we compare the contents of the idea with the nature of the symptoms. Oppenheim makes a retrograde step in accentuating the importance of reflex action as a cause of hysterical phenomena; formerly, everything that could not be readily explained was called reflex. Recent advance in knowledge is to be attributed to the recognition of the fact that the nervous processes underlying hysterical phenomena are carried on in the highest parts of the brain, in those whose action is bound up with consciousness. Unless the conception of reflex actions is unduly stretched, they cannot be called in to explain hysterical symptoms.

Every event in the world is due to change of movement in material particles. It is known, however, of some few nervous processes that the material or extrinsic side is only a part of the matter, and that some motions in the brain arise from within, or are intrinsic (will and idea). Every deeper conception of the universe recognises that this is not only true of the brain, but that all material motion is but the extrinsic expression of will and idea. If this be so every process whatever can be considered from a two-fold aspect, an extrinsic and an intrinsic one. In most things we are practically limited to the extrinsic, the intrinsic point of view being confined to our own individual internal consciousness. After long effort the identity (? concomitance) of conscious movements with material changes in the corresponding nerve centres has been established by inductive reasoning. The next step is the recognition of a corresponding inner psychical side, not only to conscious cerebral processes, but also to those which are considered as reflex. Observation fails to indicate any essential difference between psychical reaction and reflex action. Whether more or less is interposed between the afferent and efferent limbs of the reflex arc does not alter the nature of the process. The only difference is that the inner aspect of the process sometimes falls within our consciousness and sometimes does not. Every psychical process is thus a reflex action, and every reflex action a psychical process. If a distinction is sought for, then the expression reflex action must be restricted to those nervous reactions, whose intrinsic aspect remains inaccessible to cerebral consciousness as is the case in the spinal reflexes. In this sense reflex action plays but a very subordinate part in the pathogenesis of hysteria. The tendency of workers in natural science, which in general deals only with the extrinsic point of view, to fix exactly the manner of being from this aspect of that which can only be known from the intrinsic, leads to confusion and unsound hypothesis, because of the scanty knowledge of the motions of matter in the brain. Hence the inadequacy of anatomico-physiological explanations in hysteria and the erroneous explanation of hysterical paralysis by obstacles to conduction of nervous impulses.

Lastly, Dr. Möbius objects to Oppenheim's separation of the traumatic neurosis from hysteria. He thinks that the name should be retained on the ground of practical convenience, but that the traumatic neurosis is a form of hysteria differing only in degree and not in kind.

According to Dr. Cousot,[10] hysteria may show itself by an isolated symptom such as monoplegia or contracture, and this form is especially common in children. Charcot insisted that the face is never involved in hysterical paralysis, but several cases of this kind have now been observed. Hysterical anæsthesia is held to be a disorder of the personality, and the author tries to support this hypothesis by a reference to the events of every-day life. If we set about a mechanical task we go on with it unconsciously, even if a train of thought altogether foreign to it suddenly starts up and absorbs all our attention. Whilst our whole attention is fully concentrated on some fixed purpose, other events which take place in the neighbourhood may, although they pass unperceived by us, be registered in our brain. So in hysterical anæsthesia, sensory impulses passing to the brain can set free nutritive and secretory reflexes and yet not rise into consciousness. Study of hyperæthesia teaches us the meaning of many otherwise unintelligible actions of hysterical patients. Just as a patient with hyperæsthetic retina shuns the light, so hyperæsthesia of the gastric mucous membrane causes anorexia and obstinate vomiting. The onset of pains in the head, with vomiting and fever, may simulate the symptoms of meningitis, and violent pains in the abdomen with vomiting and constipation resemble those of peritonitis. Similarly the symptoms of angina pectoris and of arthralgias may occur from hyperæsthesia of the bones and joints. Dr. Cousot relates a case in which anorexia, vomiting of blood, anuria, rapid respiration (30 to the minute during a period of four weeks), successively occurred; the dyspnœa ceased during sleep. The motor disturbances in the form of paralysis, contracture or tremor, so frequently present in hysteria are attributable to the weak condition of the muscles which is indicated by the dynamometer. The soil is prepared through deficient innervation from the nervous centres; then an injury, a psychical shock or an hallucination makes the latent paralysis manifest. The tendency to contracture is an indication of the increased irritability of the pyramidal tracts, and tremor is also a consequence of this. Although hysteria is clearly a functional neurosis, yet it may affect the trophic centres. Babinski, in 1886, described muscular atrophy as present in some hysterical cases, and the author has seen a case in which muscular atrophy occurred in the region of distribution of both popliteal nerves with hysterical pains in the joints; examination under an anæsthetic showed that the joints were healthy and there was no reaction of degeneration in the muscles; the case was one of atrophic paralysis of hysterical origin. Tabes, syringomyelia, and especially paraplegia, are closely simulated by hysteria, and the diagnosis, whether the symptoms are due to organic or hysterical changes, is by no means always easy. The author concludes that the true nature of this many-sided neurosis is more likely to be reached if its explanation be sought for on the basis of known physiological laws and hysterical patients not simply dismissed as past masters in lying and dissimulation.

Experimental physiology has shown that prolonged intense or repeated stimulation of a motor centre may spread to other centres and give rise to general convulsions. Excitation of other non-motor centres may be similarly disseminated through the brain, and thus an emotion affecting first a purely psychical centre may stimulate sensory and motor ones and react on the organic functions. An emotional stimulus, by its dissemination through psychical and then through sensory and motor centres, gives rise to the series of hysterical symptoms. It is diffused in this manner because the central nervous system is over-irritable, is easily affected by suggestions of diverse kinds; this suggestibility characterising the hysterical mental state. Hence the unconscious production of a whole train of morbid phenomena, best seen and most thoroughly studied in traumatic hysteria.

II.—Papers on Miscellaneous Facts concerning, and Symptoms of, Hysteria.

Etiology of Hysteria and Neurasthenia.—Whilst all allow the influence of heredity in the causation of hysteria and neurasthenia, there is, as Dr. Löwenfeld observes, divergence of opinion as to the part played by it. At one extreme are Arndt, Charcot and the school who hold, the former for neurasthenia, and the latter for hysteria, that heredity is the only predisposing cause, all others being exciting agents (*agents provocateurs*). Arndt thinks that in neurasthenia there is a failure in the full development of the nervous system, either innate or acquired, so that the persons affected remain undeveloped or childlike in character. With this imperfect development a chlorotic blood-state is commonly present. The origin of this nervous-chlorotic constitution is derived from an inherited weakness in the progenitors, and is an expression of degeneration. The large majority of German authors see in the inherited neuropathic constitution a predisposing factor to neurasthenia which is frequently present, but is not constant, nor the only one. According to Levillain, there is an hereditary form of neurasthenia, the inherited neuropathic taint manifesting itself in certain symptoms which complicate those proper to this neurosis. The author found that seventy-five out of 100 cases of neurasthenia under his care, gave evidence of inherited nervous predisposition; in twenty-nine the mother, in six the father, and in seven cases both parents suffered from "nervousness," neurasthenia, or hysterical attacks; and in the remaining cases from other nervous disorders. He has found hereditary tendencies present in a large proportion of hysterical patients, but not in all, and that the worst forms of hysteria may be developed without any such influence. The disposition to hysteria which the brain in one case has acquired through inheritance, it acquires after birth in another through disease, such as meningitis, or through the persistent action of harmful agencies on the nervous system. How can epidemic outbreaks of hysterical symptoms, which sometimes occur amongst the people of a whole district in whom no inherited tendencies to nervous disorders have before been noted be accounted for, if the disposition to hysteria cannot be acquired? He agrees, with other writers, that in the great majority of cases of hysteria the heredity when present is a direct one. The action of the hereditary tendencies varies in particular cases remarkably. On the one hand, the children of an hysterical mother, brought up with the utmost care, and shielded from all harmful influences, early show signs of hysteria for which no exciting cause can be discovered, and on the other, patients similarly predisposed to the neurosis preserve their good health for a long time, or at the most, suffer only from certain nervous symptoms until a powerful shock or persistent depressing influence evokes hysterical symptoms.

The so-called Signs of Degeneration.—Dr. Löwenfeld thinks that too much importance must not be attached to the minor malformations, chiefly of the special sense organs and cranium, which have been described as signs of degeneration. Severe cerebral damage from hereditary taint is often shown by clear signs of bodily degeneration, but there is no constant relation between the bodily and mental anomalies. In many thus hereditarily affected in spite of evident signs of mental degeneration those of physical degeneration are absent, and *vice versâ*. According to Magnan, the characteristic features of hereditary degenerations lie in the mental state. There are the cases of agoraphobia, claustrophobia, aichmophobia, pyrophobia, folie de doute, délire du toucher, and all the group of uncontrollable impulses. Koch divides the psychopathic conditions (states of lowered capability) into three kinds; the psychopathic disposition, the psychopathic taint, and psychopathic degeneration. In hysteria and neurasthenia, the psychical changes belong to the two first or less serious conditions; but it must be borne in mind that anomalous mental states are found in neurasthenia apart from any hereditary tendencies. Arndt's view that the nature of the change in neurasthenia is essentially a want of full development of the nervous system has been mentioned, but such an arrest of development in the hereditary form of the disorder has not been made out. The author attaches more importance to variations in the development of the vessels of the brain, and therefore, in its nutrition. He points out that in no organ is the influence of nutrition, or capacity for work so clearly shown, as for instance, in the production of fatigue or exhaustion, or even loss of consciousness by hunger, hardships, over-fatigue, and loss of blood. Variations in the size of the vessels must be of importance, because they affect the blood supply, and no development of the cerebral vessels can be considered normal which does not bear a certain relation to the size of the brain. From his own observations, the author has found that the relation of the width of the arteries to the brain weight, varies very considerably. Physiological and pathological observations show that for the recuperative power or elasticity, which is so important a property of nerve-cells, the condition of nutrient supply is of the greatest importance. Defective development of the vessels of the brain entails a predisposition to all the disorders produced by causes which exhaust the nerve-centres, and interfere with brain-nutrition.

Influence of Sex.—The proportion of men to women affected with hysteria is put at 1 to 17 in Germany, whereas Gilles de la Tourette, in Paris, and Pitres, in Bordeaux, found it to be 1 to 3, so that the conclusion is that in the lower classes from which both series of statistics were drawn the disposition of men to hysteria is much greater in Paris and in Bordeaux than in Germany. In the better classes frequenting health resorts, the author found the proportion of male to female cases to be 1 to 6, and in childhood 1 to 2. The author endorses the commonly received view that the mental characteristics which distinguish women from men—the predominance of the emotions over the intellect—is the foundation of the greater tendency of women to suffer from hysteria.

As regards neurasthenia, Beard and Bouveret think that it is more frequent in men, Möbius in women, and Levillain that both sexes suffer equally. The author's own observations have been that most men suffer, but he thinks this may be partly accounted for by the fact that so many women with nervous troubles consult the gynæcologist and not the physician.

As to occupation, neurasthenia is especially frequent in those who work with their brains rather than with their hands, particularly if the occupation yields a slender and precarious means of subsistence, with much anxiety, responsibility, or interference with proper rest. Hysteria does not especially affect this class. The author cannot confirm Gilles de la Tourette's statement that hysteria is most frequent in those of the labouring class whose occupation exposes them to accident, and hence to traumatic hysteria; he thinks that workmen who have received injuries to their nervous systems suffer from neurasthenic conditions, and not especially frequently from hysteria.

Race and climate have no especial influence.

Exciting Causes—Psychical Overwork.—Arndt puts the question in another point of view from that generally stated, namely, that neurasthenia is produced in very many cases, not by mental work absolutely excessive in itself, but by a relative incompetence to fulfil the daily task. The author distinguishes three groups of cases in which excessive brain work acts as the cause of neurasthenia and hysteria.

(1) Those persons who have undoubtedly fallen ill by reason of their daily work, although they have not worked extraordinarily hard, but have solely performed the average duties of their place, or even less than these, form a great many of the cases. Examination candidates who break down after a course of study of only average severity afford an example. Again, in our times the ambition of parents for their children to rise out of their own class makes them put the children to unsuitable occupations, and thus many persons who would have remained healthy at mechanical trades, when put to continuous sedentary occupations involving brain-work, break down because they are not fitted for them.

(2) The second group comprises persons who have undoubtedly worked excessively hard, but even in them the nervous exhaustion is not to be entirely attributed to overwork, but is also due

to concomitant emotional disturbances of a depressing character, *e.g.*, hard work, with uncertainty of making a livelihood by it; the being superseded by a junior after years of hard work.

(3) The smallest class consists of those who suffer simply as a consequence of excessive brain-work.

Overpressure at School.—The author does not agree with Charcot's view that it is impossible to overwork school children, because when the limit of their brain power is reached they simply give up, and cannot be urged on to make fresh exertion with a tired brain as their elders can, but, on the contrary, thinks that in children it is just as easy to establish a condition in which by an effort of will the over-fatigued brain permits an attempt at work beyond its powers. Hertel, in Copenhagen, found seven per cent. more invalids amongst the children of a school with the longest hours of work than amongst those who worked for shorter times. Axel Key, in Stockholm, found five per cent. more sickness in the first than in the second class. With regard to the question of how far there may be overpressure in the system of education in the German higher schools (Realschulen, Gymnasien, Tochterschulen), the author says that for half of the higher classes there is certainly possibility of excessive mental strain, and for the other half overpressure may be indirectly occasioned by the lack of sufficient time and opportunity for the care of the body and physical exercise. Neglect of fresh air and exercise is most injurious, the mind being developed at the expense of the body. This want of care for the physical health and excessive stimulation of the brain is especially hurtful, because a large proportion of the children frequenting these schools have inherited a disposition to nervous troubles. Muscular over-exertion seldom plays the part of sole exciting agent of hysteria; mostly it acts in association with emotional disturbance or other injurious influences.

Erb,[11] in a speech "On the Increasing Nervousness of our Times," defines this nervousness as a condition of weakness, exhaustion, and increased irritability or a morbid increase and persistence of the physiological processes of fatigue. Neurasthenia, the most common and important form of this nervousness, consists of increased irritability with great weakness, sense of fatigue and exhaustion on the one hand, and diminished powers of work on the other.

He thinks there has been during the last century an undoubted increase of the sufferers from neuroses, in neurasthenia affecting all classes of workers, especially brain workers, and in hysteria in the male subject. Any cause which overtaxes, strains and exhausts the nervous system may bring on the diseased state which is to be regarded as a pathological persistence of fatigue (exhaustion). Neurasthenia is a disease of brain workers, hysteria is a result of over-stimulation of the emotions and sensations, but there is no sharp line of demarcation between them. The causes are overwork at school in ill-ventilated class-room, and in preparation for examinations; overwork in occupations arising from the long hours of work necessitated by competition encroaching on the time necessary for rest and recuperation. Many states of nervous weakness result from overstrain on the emotional side, anxiety, cares, fears, disappointed hopes, speculation, religious, social, and political struggles.

The physical causes are muscular overstrain, sexual excesses and aberrations, especially masturbation, abuse of alcohol, tea, coffee and tobacco; severe accidents, especially railway accidents, effects of fevers, influenza and diseases of the blood.

Most important is the nervous disposition (neuropathic) which may be inherited or acquired through any of the causes above mentioned; this forms the basis on which the exciting causes work. Consideration of the causes explains the frequency of neurasthenia in the upper classes, in brain workers, in men more than women, and of hysteria in women rather than in men. The excessive tendency to the neuroses in the Latin peoples, in the Jews, from their mode of life and consanguineous marriages through long centuries, and in the Americans from the life of overstrain they lead, can thus be understood.

Affections of Sexual Organs.—The relations between diseases of the sexual apparatus in women and neurasthenia and hysteria Dr. Löwenfeld sums up under four heads.

(1) The affection of the sexual organs, which may be serious disease, or such disorders as flexions of the uterus, only give opportunity for the occurrence of nervous disturbances. The sexual affection in the majority of these cases acts on the basis of a neuropathic predisposition.

(2) The disease of the sexual organs acts in combination with a number of other injurious influences in inducing nervous disorders.

(3) Sexual affections and nervous troubles arise independently of one another, but are coincident.

(4) The sexual affection is the consequence of nervous disorders primarily arising from other causes, and may then itself lead to an aggravation of the nerve troubles.

In men, diseases and disorders of the sexual organs give occasion for the onset of neurasthenical and hystero-neurasthenical (rarely purely hysterical) symptoms. This seems to occur only in those pre-disposed to neurasthenia. Gonorrhœa and the various inflammatory (acute and chronic) affections of the sexual apparatus which form its sequelæ are the most important diseases in this respect.

With regard to sexual excesses as causes of neurasthenia, the author concurs in the views commonly received.

Other causes of neurasthenia are various forms of dyspepsia enteroptosis of Glénard, possibly nasal affections, errors of accommodation insufficiently corrected, any affection which gives rise to lasting pain. Of organic nervous diseases tabes dorsalis, chronic transverse myelitis, and spastic paraplegia are most often connected with neurasthenia, whilst the association of multiple sclerosis with hysteria is, as has been shown by Buzzard's researches, very frequent.

The acute diseases, such as enteric fever, may be followed by neurasthenia as a consequence of the interference with general nutrition, and of the weakness thus induced; but in the case of influenza, the influence of the specific poison is admitted as directly causing neurasthenia, or hystero-neurasthenical conditions which may last for months or years. Influenza is singular in this respect, as in none of the other acute specific diseases, and the same applies to syphilis, has the author been able to find clear evidence of a direct action of the specific poison of the disease upon the nervous system.

Uric Acid Diathesis.—Ebstein states that no small number of so-called neurasthenics are gouty persons in whom sooner or later the true nature of their disease declares itself. The author thinks that the uric acid diathesis plays an important part amongst the causes of neurasthenia. He also gives a warning against the incautious employment of measures directed for the relief of obesity. In several cases treatment for this end has caused severe neurasthenia.

SYMPTOMS OF HYSTERIA.

Hysterical symptoms are considered by Löwenfeld under two heads: (1) the stigmata; (2) accessory symptoms. The author thinks that the stigmata are relatively more infrequent in South Germany, especially hemianæsthesia, extensive anæsthesia, and hysterogenic zones, both in private patients whose cases cannot always be fully investigated, and in hospital ones.

Hückel is also of the opinion that hemianæsthesia is more common and better marked in France than in England or Germany, and that hysterical paralyses are more rare in Germany than in France. In Tübingen where hysteria is common and not only in mild forms, hemianæsthesia, "ovarie" and hysterogenic zones are rare.

Hysterical Anæsthesia.—As to the origin of hysterical anæsthesia, its greater frequency in some groups of cases than in others shows the importance of the patient's surroundings, *e.g.*, the symptoms presented by his neighbour in hospital. That hysterical anæsthesia is more common in France than in Germany is partly due to unconscious imitation. An hysteric who sees and hears in a hospital that hemianæsthesia is a common symptom in this disease, and further notices that the doctors take a great interest in its occurrence is sooner or later likely to develop the symptom. Other hysterical symptoms, convulsions, mutism, aphasia, so often arise in similar ways that it would be strange if anæsthesia did not also do so. Prolonged physical examination may have a similar influence in bringing out or fixing anæsthesia. Difference of duration of anæsthesia in different patients may partly be accounted for in this way. The action of the so-called æsthesiogenic agents partly depends on suggestion, but nevertheless some obscure physical action of these agents cannot be excluded, for exact imitations of magnets have no effect when true magnets produce an æsthesiogenic influence.

So far Dr. Löwenfeld has not been able to discover hysterogenic zones in the different hysterical neuralgias which he has observed.

Ovarie.—Is the ovary really felt in the "ovarian zone"? The author says that in hysterical and non-hysterical women, with thin abdominal walls, he is certain that in the spot indicated by Charcot, the ovary can be felt, and gives rise to a special feeling of pressure which is not given by surrounding parts. At the same time, he does not mean to say that the painful feelings on pressure always come from the ovary itself; the presence of hyperæsthetic areas in the same situation in men contradicts this.

Hysterogenic Zones.—In discussing Bernheim's view that hysterogenic zones are due to suggestion, and that no influence can be supposed to come from the hysterogenic spots themselves, he states his opinion that, although a great number of the zones found by French authors rest on a basis of suggestion, yet this is not true for all of them, and gives cases in which the hysterogenic influence was not due to suggestion. The extraordinary readiness with which in the patients in the Salpêtrière, and at Bordeaux, hysterical attacks were excited from the most diverse parts of the body (whether these were anæsthetic or hyperæsthetic) appears to him to be connected with suggestive influence. From a practical point of view these zones have not the importance French authors attribute to them; and in oppositon to Pitres, he thinks that they can only very seldom be of therapeutic avail in arresting an attack.

Hæmoptysis in Hysteria.—Hæmorrhage from the air-passages is rare in hysteria, but may be observed in men as well as in women. In the latter, it may be vicarious of menstruation, in which case there is generally little cough. In one male case, reported by Laurent, it never occurred except during an hysterical attack. E. Wagner has met with a sputum of exceptional characters, coming probably from the mouth, in a number of badly-nourished hysterical patients with cough; it lasted weeks or months, and excited suspicion of tuberculosis, although physical examination excluded disease of the lungs and bronchi. The sputum was from 20-100 grammes in quantity in the twenty-four hours, was reddish but not rusty; it contained numerous small grey particles and blood-cells, but no formed elements from the lungs or bronchi.

Circulatory Disturbances.—A feeling of heat in localised areas accompanied by reddening of the skin, and objective rise of temperature of the part, is not uncommon, especially in the face. Brodie observed that in arthralgia first came coldness and pallor, followed after some hours by redness, increased heat, and sweating of the part.

In a patient of Rosenthal's (a girl of 23), discolouration of the hands with feeling of cold preceded hysterical attacks. The temperature of the hands on days free from the attacks, 34·4° C. fell to 30·6° C. before the attack; they became pale, the finger tips and nails blue. Afterwards the temperature rose to 35·6° C. with red fingers and nails.

Hysterical Hæmatemesis.—In hysteria, hæmatemesis may be vicarious of menstruation, but often occurs quite independently of or during the menstrual periods. Certain patients are taken suddenly with hæmatemesis in relatively good health and with almost complete absence of pain. In other cases it comes on amongst other symptoms of the neurosis, in the course of grave hysterical affections. In many patients, burning or gnawing pains, with exquisite tenderness in the epigastrium, occur, and vomiting of blood is attended with increased pain, anxiety, and sometimes a faint of short duration. In the majority the hæmorrhage recurs, sometimes daily; the quantity of blood in twenty-four hours may amount to a litre, and there may be melæna. Severe anæmia and weakness are thus caused. The author, after the above account, says that the diagnosis from gastric ulcer can only be made with confidence in cases in which hæmatemesis occurs without long preceding gastralgia and dyspepsia, or in which it follows other hysterical hæmorrhages, and that in view of the frequency of ulcer in young women, the presence of an ulcer should be first thought of in diagnosis. The writer thinks that seeing the obscurity of symptoms of gastric ulcer in many cases, too great caution can hardly be exercised in diagnosing hæmatemesis as of hysterical origin.

Hysterical Œdema.—Hysterical œdema, as Sydenham described it, is most marked in the evening, does not pit on pressure, is seldom an isolated symptom, but accompanies paralysis, contracture or arthralgia. It is generally confined to one foot, hand or limb, but may affect more than one limb at the same time. In some cases the skin over the swelling is normal in colour, in others it is cyanotic or even dark blue; Charcot has produced this œdema by suggestion. A lowering of the temperature generally accompanies the œdema. The development and decline is characteristically rapid. The duration depends on that of the motor disturbances accompanying it. Striking changes in the swelling may occur during the menstrual periods or under emotional excitement.

Muscular Atrophy.—It is now known, in opposition to former views, that muscular atrophy may occur in hysteria. Dr. Lowenfeld classifies the cases as follows:—

(1) Simple atrophy.

(*a*) In connection with paralyses and contractures. It may develop very quickly. No fibrillary contractions nor R.D. Electrical irritability falls proportionately to the muscular wasting.

(*b*) Unconnected with paralysis or contracture (isolated affection). Gilles de la Tourette and Dutil say that the atrophy occurs most frequently on the side of the seat of other hysterical symptoms, especially of disturbances of sensation.

(2) Degenerative atrophy, with fibrillary contractions and R.D.

The actual occurrence of muscular atrophy in hysteria is no longer open to doubt. In the diagnosis from atrophies of organic origin, the degree and extent of atrophy, loss of electrical irritability and presence of fibrillary contractions give no aid. The development of the hysterical form is somewhat characteristic. It comes on remarkably quickly, and reaches a severe degree in a short time. The further course is variable. In many cases it quickly gets better, in others it lasts long, sometimes even for years, stationary. Observations do not yet show whether recovery takes place in all cases, or whether the cases of atrophy with R.D. follow a less favourable course than those of simple atrophy. Of greater weight is the relation of atrophy to other hysterical symptoms. It may almost be said that it exclusively affects parts which are or have been the seat of other hysterical troubles (paralysis, contracture, anæsthesia).

Aphasia.—The author thinks that, in addition to mutism, true aphasia may occur in hysteria, though most cases described as hysterical aphasia are in reality mutism. The author gives a case from his own practice in which motor aphasia followed a severe hysterical convulsion in a girl of 24, and quotes two others, one from Charcot,[12] and another reported by Dobie.[13]

Convulsive Attacks of Hysteria (Löwenfeld).—In answering the question what symptoms are characteristic of the hysterical fit, similar difficulties are presented as in the case of epilepsy. Convulsions are the most striking characteristic of the ordinary epileptic fit, but they occur in very variable degree and may be absent. Paroxysmal loss of consciousness has, therefore, been selected as the essential feature of the fit, but is not an absolute criterion, since epileptic fits may occur without it. In the great majority of hysterical attacks the striking feature is the convulsions, but they may be inconspicuous or absent, and a characteristic must be found which is present in every attack, with or without convulsions. The author selects a certain morbid mental change as this characteristic, and this may vary in intensity from a slight clouding over of perception to the full interruption of the normal stream of consciousness, to the entrance into a psychical state wholly different from the normal (double consciousness), or to apparently complete loss of consciousness. This mental change, however, may be absent in hysteria, just as loss of consciousness may not occur in epilepsy.

The succession of the typical stages of the attack as described by Richet is seldom met with. Single periods of the complete hysterical attack are observed in all their details as at the Salpêtrière, but isolated or joined to one another in altogether irregular ways and in very various degrees of development. This atypical succession of the different stages of the attack is the most common form. The fully-developed "*grande attaque*" is thus rare in all countries, but variations or modifications of it are often met with, due to the occurrence of one of the periods as an isolated attack. One variety is the isolated occurrence of the epileptoid stage, which may be very difficult to distinguish from true epilepsy. It was formerly believed that hysteria through increasing severity of attacks might later develop into epilepsy, and also that there was a mixed form of attack intermediate between hysteria and epilepsy. But we now know that hysteria and epilepsy are two neuroses, which neither pass into one another nor in any way form a conjoint affection. Nevertheless, hysteria and epilepsy may exist side by side in the same case. Generally, epilepsy is the primary affection. After epilepsy has been long present accidental causes lead to the development of various hysterical symptoms, either of the persistent or of the paroxystic kind.

Hysterical Pyrexia.—In a valuable paper, Dr. Sarbo[14] says that Briquet distinguished three kinds of hysterical fever. (1) Symptoms characterised by the quick pulse, that accompanied them. (2) Quick pulse and subjective feeling of heat without other symptoms. (3) In addition to the above-named symptoms, pains in the head, feeling of thirst, anorexia, faintness, &c.

Bouchut recognised only the form of hysteria with attacks, and distinguished it as a paroxysmal neurosis free from fever, from another neurosis in which fever was present, and which he named "chronic neurasthenia." But this latter is merely a variety of hysteria, and his classification has not been accepted. Under the name of "hysterical fever of pseudo-meningitic type," Boissard,[15] Chantemesse, and others describe a new form. The name of this affection sufficiently describes the symptoms.

Gagey[16] divided hysterical fever into three forms: (1) continuous; (2) remittent; (3) intermittent: the latter may assume a quotidian or tertian type. His opinion was that an hysterical fever exists, of the nature of which we are ignorant, and which often comes on after childbirth, severe illnesses and psychical disturbances; the severity of the symptoms is out of proportion to the elevation of temperature.

Brian[17] recognises three forms of hysterical fever: (1) Chronic, lasting a long time; an irregular febrile condition in which the elevation of temperature does not correspond to the severity of other symptoms; this form corresponds to Gagey's first division. (2) Of short duration, like typhoid, which attends the onset of hysteria, and in which the temperature reaches 39°-39.5° C. (3) Intermittent, generally assuming the tertian type. He cites several other authors as also describing an intermittent type.

Fabre,[18] who considers hysteria as a disease of the vegetative organs *par excellence,* distinguishes five forms of hysterical fever: (1) ephemeral; (2) chronic; (3) intermittent; (4) typhoid; (5) hysterical febricula. This author also describes cases of hysteria, in which the symptoms predominate in the lungs, and simulate pulmonary phthisis.

Pinard opposes the views of the above writers; he doubts whether there was actually any fever, points out that the amount of urea was not estimated and that no loss of weight is mentioned, which would surely have occurred if there had been fever. Some cases he thinks were abortive typhoid. In others, symptoms were present, resembling those of fever, but in which there was no rise of temperature, and which he calls therefore "hysterical pseudo-fever."

Later, Chauveau distinguished: (1) A slow form, a long lasting condition of moderate fever (38-39° C.) without loss of weight. (2) Intermittent form. Most cases described under this head Chauveau regards as cases of true intermittent fever, but he thinks that hysterical cases also occur. (3) Form of short duration. The pseudo-meningitic type he refers to his second variety. He ascribes the pathogenesis of hysterical fever directly to an irregular action of the heat centre. Debove succeeded by means of suggestion in raising the temperature to 38·5° C.

Of German authors, Wunderlich[19] especially drew attention to the temperature alterations to be observed in hysteria. He emphasises the occurrence in weakly persons, and in nervous persons of hysterical constitution, very often on the slightest provocation, or without any at all, of elevations of temperature which may last a long time. Sometimes the remarkable phenomena of a different temperature on the two sides of the body is noticed. Dr. Sarbo observed several times an elevated temperature on one side of the body, without any organic change in one or other side, in persons suffering from hysteria, or spinal affections.

Rosenthal[20] mentions in severe hysterical conditions local redness of regions of the skin and elevation of temperature with a pulse of 100-130, and an axillary temperature of 37·4-37·6° C. Rosenthal thinks that febrile symptoms may be present in hysteria, but true fever, *i.e.*, an elevation of the body temperature, never. Hysterical fever is, therefore, a misnomer, and leads to confusion.

Hale White[21] gives the chief features of hysterical pyrexia as follows: (1) it always occurs in girls; (2) they are often otherwise hysterical; (3) they are always of that age at which hysteria is most common; (4) ovarian pain and tenderness is often present; (5) rigors may be present; (6) delirium, rapid pulse, flushings, or a trace of albumen in the urine, are often found; but any one or all may be absent; (7) the characteristics of the temperature are its erratic behaviour, both diurnally and generally often varying in different parts of the body, and quite unlike the temperature of any known inflammation or fever, as well as the occasional great height which it may attain.

Moravcsik[23] gives the following summary of his observations: (1) In the hysterical, the body temperature, pulse, and respiration show fluctuations. (2) A rise of temperature occurs without any ascertainable organic changes. (3) The height of the temperature often bears no relation to the rate of the pulse and respiration. (4) The morning temperature is often higher than the evening. (5) The pulse and respiration show the greatest fluctuations. (6) Increased rapidity of pulse and respiration rate often precede a convulsion. Moravcsik also observed an elevation of temperature produced through suggestion during hypnosis; as did also Krafft-Ebing.

A fever, of allied nature, is found in the high temperatures which occur after the status epilepticus. By Charcot and his school, this fever was considered to be due to muscular overwork. Witkowski considers that the rise of temperature is not due to muscular work, but that there is some connection between it and loss of consciousness. He entitles the fever which occurs in general paralysis, uræmia and disseminated sclerosis with loss of consciousness, "sensory fever." He considers hystero-epilepsy as a variety of epilepsy, in which view Dr. Sarbo does not follow him, preferring, with the French school, to regard hystero-epilepsy as distinct from epilepsy, and the fever observed to occur in it as an "hysterical fever."

Its Pathogenesis.—Dr. Sarbo then gives an abstract of all the chief papers, from Virchow's downwards, on the regulation of the temperature by nervous heat centres, and sums up the most important points as follows:—(1) Clinical observations indicate that the central nervous system plays a part in the origin of fever and the discovery of the vasomotor and thermal centres by experimental research confirms this. (2) Those fevers, which have no substratum of organic disease, and are therefore conditioned by the nervous system alone, are to be considered as originating in disturbance of the function of the thermal centres, and may be named "functional nervous fever." (3) It is a *priori* probable that in hysteria, the functional neurosis *par excellence*, such fever should occur.

The author divides cases of hysterical fever into two groups :—

(1) Those described as hysterical fever, but in which no thermometric measurements were taken.

(2) Those in which the temperature was taken comprise two sub-varieties: (*a*) Those in which there was no rise of temperature (chiefly author's own observations). (*b*) Those in which a rise of temperature was present (his own and other reported cases.)

(2a) Hysterical pseudo-fever, hysterical tachycardia. In these cases there are the symptoms of fever, headache, rapid pulse, thirst, dizziness of head, &c., but no rise of temperature. The author draws the following conclusions from six cases of his own and one of Rosenthal's of this nature.

(1) Hysteria as a vasomotor neurosis may show a group of symptoms, which, although tachycardia is associated with them, give the impression of a febrile condition; since, however, there is no actual rise of temperature, he calls this condition "hysterical pseudo-fever" although he does not include all the cases included by Pinard under this term (Pinard reckoned cases with a temperature of 38-38·5° under this category, considering this temperature physiological).

(2) Hysterical tachycardia is a symptom pretty often observed.

(3) This pseudo-fever appears in simple hysteria as well as in hystero-epilepsy.

(2b) *Cases with actual rise of Temperature.*—The author has endeavoured to make a more satisfactory classification than those hitherto proposed which shall keep in view the pathogenesis of the condition and its analogy with other hysterical phenomena. He divides them into two main classes (1) continued fever; (2) attacks of fever. Both these varieties can be sub-divided into a severe and mild form according to the height of fever. The continued fever has an analogue in the sensory sphere in the hysterical anæsthesias of long duration, and in the "état de mal" several times observed in hysterical seizures.

Similarly the attacks of fever may be compared with quickly passing anæsthesias, and with single convulsions.

Hysteria
- Anomalies of thermal centres (*a*) continued fever (*b*) attacks of fever —severe above 38·0° C.; mild below 38·5°.
- „ „ Sensibility (*a*) persistent anæsthesia (*b*) ephemeral anæsthesia—either may be superficial or deep.
- „ „ Motion (*a*) "état de mal" (*b*) isolated convulsive attacks—either may be severe or mild.

These resemblances are justified by the connections observed in his cases between the convulsive attacks and the febrile states, and in the correspondence from the point of view of the variability of these states and the other hysterical symptoms.

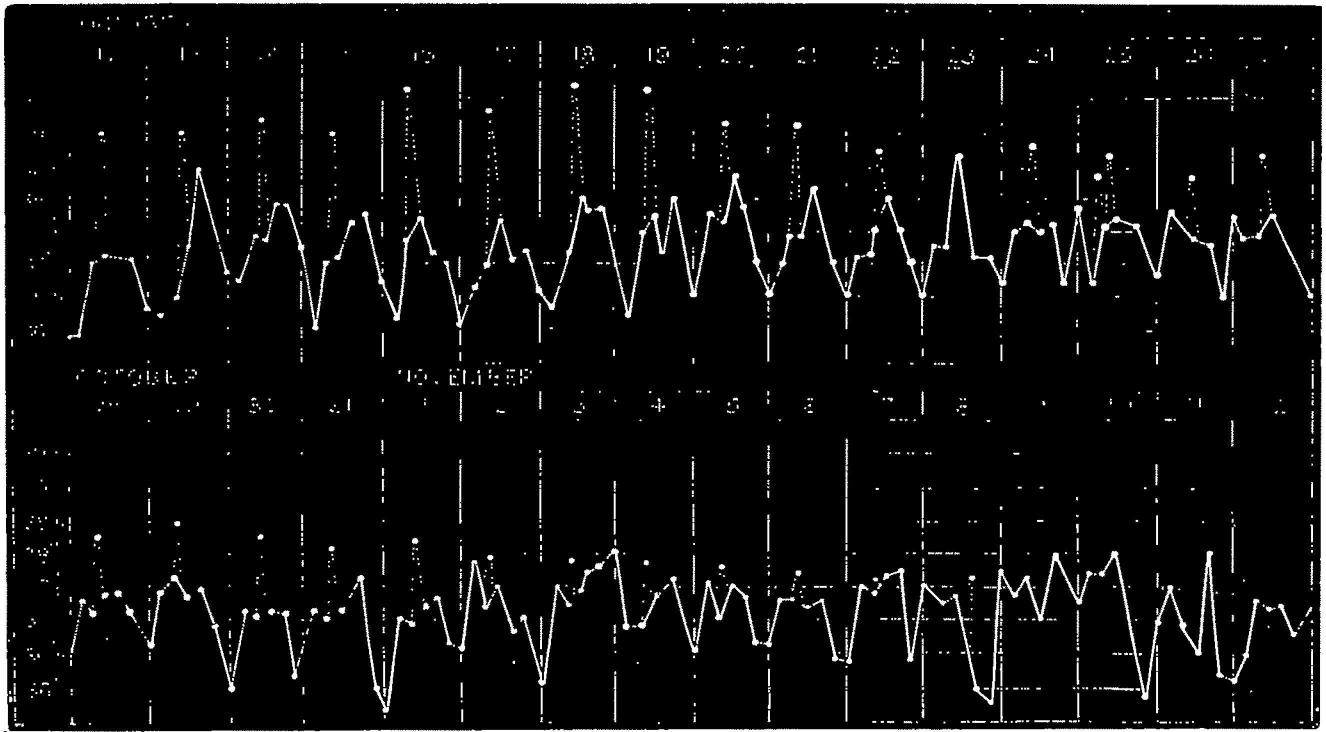
The author cannot agree with Pinard that a temperature of 38°-38·5° C. is physiological; he found it present only exceptionally, in the course of observations taken six times a day for over seven months at a time, and then only in connection with other febrile symptoms, the normal temperature being 37·1°. He considers that this indicates a less degree of the same changes that take place in the heat centres when a higher temperature is produced, as for instance in the case of tuberculosis where a feverish condition is generally spoken of with a temperature of only 38 or 38·5° C.

A. *Hysterical continued Fever*: (1) *Cases with slight rise of temperature.*—Dr. Sarbo discusses some cases at length, and then criticises Charcot's dictum that as a rise of temperature never follows an hysterical whilst it may be present at the close of an epileptic attack, a rise of temperature in any case of convulsions indicates their epileptic nature. This diagnostic aid loses its value, because after an hysterical "état de mal," fever may come on. He has observed a case of undoubted hystero-epilepsy in which, after a severe convulsive attack, a paroxysm of fever came on, and this was not connected with the convulsion, as fever subsequently was present without convulsions. Cases are met with in which hystero-epileptic convulsions are combined with hysterical fever.

In this group are comprised the cases of fever with symptoms resembling meningitis (*fièvre pseudo-méningitique*). He thus sums up the characteristics of the group:—

(1) In the course of hysteria, continued fever, not dependent on organic disease, may appear; it is not to be considered as due to muscular work (in the convulsive attacks), and on account of its dependence on hysteria alone should be called "hysterical continued fever."

(2) Hysterical continued fever can be divided into two grades, according to the elevation of temperature, whether below or above 38·5° C. Its duration varies from days to months.


18
19
20
21
22
23
24
25
26
OCTOBER
NOVEMBER
30
31
1
2
3
4
5
6
8

(3) The fever has no settled type. It may appear and disappear quite suddenly.

(4) Such anomalies as a difference of temperature on the two sides of the body, and a low evening and high morning temperature, are met with.

(5) The severity of the accompanying symptoms often bears no relation to the slight rise of temperature.

(6) The fever may be combined with such a group of symptoms, as to resemble the picture of a known disease, *e.g.*, typhoid, tubercular meningitis, phthisis, and peritonitis. On account of the accidental character of these groups of symptoms, the division of the cases into sub-varieties according to the symptoms present, as French authors have done, is not desirable.

(7) In most cases no cause for the fever is discoverable; in some stoppage of menstruation, psychical shocks, and injuries appear to be causes.

(8) In some cases this hysterical fever comes on after, but is independent of a series of convulsive attacks (ètat de mal hystèrique). Accordingly the onset of high fever is no criterion in the distinction between hysterial and epileptic convulsions.

(9) This form of hysterical fever has so far been observed in the majority of cases of hystero-epilepsy.

B. *Hysterical paroxysmal fever* (attacks of fever), (1) attacks of mild type.

The author reports an instânce in a woman of 24 years of age. She suffered from hysterical pains in the abdomen, but neither these nor the retention of urine, constipation, and meteorismus from which she suffered at times were connected with the attacks of fever, in which the temperature rose to 38° C. Fever came on from time to time and lasted for an hour or two, and was attended with accleration of pulse and respiration.

(2) *Attacks of High Fever.*—Case of hystero-epilepsy; the patient, a girl of 16, suffered from severe hystero-epileptic attacks, originally brought on by a fright; from anæsthesia of the right side of the body, right amblyopia with contraction of visual fields. Loss of voluntary movement in the right extremities; the temperature was reduced over the right side of the body. The last attack occurred three weeks before admission, and left a pain in the head. A chart of the temperature is appended. Malaria, typhoid and tuberculosis could all be excluded as causes of the fever, and no organic disease was discovered; the diagnosis of hysterical fever was therefore made by exclusion. The attacks

of neuralgia from which the patient suffered were so changeable in character that they could have had no causal connection with the febrile paroxysms. The rise of temperature came on daily almost at the same hour, with feelings of heat, apathy, somnolence, flushing of skin, which was hot to the touch, rapid pulse and thirst, ended in profuse sweating, and the patient fell into a deep sleep and awoke with headache. The temperature on the right (anæsthetic) side of the body was ·3-·5° lower than on the other side; the author has observed this lowering of temperature on the anæsthetic side in another case, and quotes similar observations of Rosenthal.

Conclusions from consideration of cases of hysterical paroxysmal fever:—

(1) In the course of hysteria, attacks of fever sometimes occur without assignable reason.

(2) These febrile attacks are attended, according to their intensity, by moderate elevation of temperature.

(3) In many cases the fever accompanies a number of severe symptoms which are incidental, and have no common origin with the elevation of temperature.

(4) Attacks of fever may be combined with, but are not dependent upon, convulsive attacks.

(5) "Hysterical pseudo-fever" as well as "hysterical continued fever" may be combined with this form.

(6) This form occurs both in simple hysteria and in hystero-epilepsy.

Ætiology.—Hysterical fever is to be considered as a symptom of this functional neurosis, and any cause which can evoke hysteria can produce hysterical fever.

To sum up—

(1) There is a true hysterical fever which occurs in two forms: (1) continuous; (2) paroxysmal.

(2) Hysterical is to be considered as a functional fever, and a phenomenon analagous to other hysterical symptoms.

(3) It may occur either in simple hysteria or in hystero-epilepsy,

Hysterical Polyuria.—Dr. Albert Mathieu[23] observes that hysterical polyuria is not a very rare affection; it generally affects men. As a rule, the quantity of urine passed is from four or five to seven or eight litres daily, but it may reach eight or ten litres. There is more or less thirst, according to the amount of urine passed. After the polyuria has lasted some time the patients suffer from weakness, partly arising from disturbed sleep, from anæmia, and loss of flesh. There is polyphagia, but not so marked as in diabetes mellitus. Lately, the author has come across two cases in which larger quantities of urine were passed. A young man of 25, in the Hospital Hôtel Dieu, passed 25-30 litres of urine in twenty-four hours. The quantity was some time after reduced to three litres on M. Thiroloix's giving him some cachets of common salt, having succeeded in persuading him that it was a powerful remedy against polyuria. This cure by suggestion is the more interesting on account of the patient presenting no signs of hysteria.

Another man of 30, in the Hospital Tenon, whom M. Babinski had cured, by suggestion, the year before of paralysis of the radial nerve, was affected with polyuria after a blow on the head in a fight. He passed 20-25 litres of urine daily, and the author was able to reduce the polyuria momentarily, by suggestion during hypnotic sleep. There are, therefore, two types of the disease; in the first, the average quantity of urine per diem is 5-6 litres; in the second it is 20-30 litres.

M. Ehrhardt finds that the urine is pale in colour in these cases and contains no albumen or sugar. The amount of phosphoric acid is normal; that of chloride of sodium is increased in proportion to the quantity of urine passed. The amount of urea is normal or increased, according as polyphagia is present or not. As polyphagia is generally present, the quantity of urea is generally increased.

The reasons which lead to the conclusion that a given case of polyuria is hysterical in nature are: (1) its coincidence with other hysterical phenomena; (2) the mode of onset; (3) its disappearance and re-appearance under the influence of suggestion.

Under (2) polyuria may result from a strong emotion, a great disappointment, and violent injuries, especially to the head. M. Ehrhardt saw it occur twice after the ingestion of iced water during sweating; and after copious libations. It is certain that many cases of diabetes insipidus occurring after physical or mental shock are really dependent on hysteria; the causes enumerated above are the common ones of diabetes insipidus, and together with the experiment of Cl. Bernard, who produced polyuria by lesions of the fourth ventricle, show the neuropathic origin of the disease.

From the point of treatment, it is important to recognise the influence of suggestion in various forms. Sedatives such as opium, belladonna, valerian and bromide have had good effects. We have seen that small doses of chloride of sodium brought about a remarkable cure. But whatever drug is ordered, the physician must have the air of the greatest certainty as to its efficacy. Valerian and bromide should be given in large doses, accompanied by another element in massive doses, that is, suggestion.

Dr. Tenien[24] gives a full account of a number of interesting cases of hysteria. His views on hypnosis are that it is useful only in hysteria, that cases cured by it are hysterical, and that if a case of apparently organic disease is relieved, this is due to the coincidence of hysterical symptoms.

He attributes the frequency of hysteria in La Vendèe, relatively to other country districts very great, in part to the frequent consanguineous marriages.

Mixed Forms of Hysteria and Neurasthenia (Löwenfeld).—(1) Cases of hysterical or hystero-neurasthenia. Typical neurasthenic condition, generally of cerebral or cerebral-spinal form, together with single and generally slight hysterical symptoms. A special form is an hereditary one in which different anxious states occur, *e.g.*, agoraphobia and temporary spasms of the pharynx of such obstinacy as to render the taking of nourishment difficult.

(2) Cases in which signs of neurasthenia first appear, and then those of hysteria show themselves and proceed to greater relative development; the latter may be ushered in by a hysterical attack. In many cases a hypochondriacal or melancholic temperament is present. This condition has been considered of especial importance in the traumatic neuroses, but may occur in cases of non-traumatic origin.

(3) In which hysterical symptoms predominate, but for a longer or shorter time retire into the background and the signs of neurasthenia predominate.

[Dr. Shaw and Dr. Duryea ("Œdème bleu des hystériques"[24a]) report the case of a young woman who received a blow on her left breast whilst being rescued from drowning, and a few days after began to have convulsions. The seizures averaged two a week for a time, during which the left breast increased in size and became so painful that an incision was made into it, but no pus found. In April, 1892, the breast was enlarged, somewhat pendulous, soft, œdematous, rather cyanotic and intensely hyperæsthetic. There was blue œdema of the left hand and forearm, with coldness. The hyperæsthesia extended from below the breast to the shoulder, and a similar area existed over the left scapular region. There were also considerable areas of anæsthesia on the left side, limited to the anterior aspect of the extremities.

She had numerous convulsive attacks and other hysterical manifestations. The authors remark that œdema, as a symptom of hysteria, although it has been mentioned by a number of authors from Sydenham downwards, very rarely comes under the observation of the profession at large.]

III. Papers on Hysterical and some other Functional Disorders of Motion.

Hysterical Paraplegia.—Dr. Lowenfeld, on the diagnosis of hysterical paraplegia, which often presents considerable difficulty, says that the first thing to ascertain is the presence of actual paralysis. The condition may be one like that of astasia-abasia, or like that found in neurasthenia, in which the patients are afraid to walk because of the pains or disagreeable sensations caused thereby. Further examination may show that loss of power is not present to the extent expected from the patient's statements and behaviour, and with some insistence on the physician's part they may be able to stand and even to walk when they believed that they could not do so.

If paralysis is actually present, the frequent combination of hysterical with organic nervous disease, especially in spinal cord affections, must be borne in mind. The history of the case is important, and it must be remembered that a sudden onset often occurs in myelitis. Pains and paræsthesiæ are common in organic, rare in hysterical paraplegia. Anæsthesia of decided hysterical character may be found. The condition of the knee-jerks, which are very seldom absent in hysteria, is of more importance than that of the plantar reflexes. Escape of the bladder and rectal functions in the presence of severe paraplegia, is against organic disease.

With regard to the supervention of organic charges on long-lasting hysterical contracture, the author remarks that in Charcot's celebrated case in which sclerosis of both pyramidal tracts was found at the autopsy of a patient who had suffered for many years from hysterical spasmodic paraplegia, that this may have been an instance of the frequent combination of hysterical with organic affection of the spinal cord.

Hysterical paralysis of pyschical origin may be due :—

(1) To the non-representation in the cortex of movements of affected parts, so that the movements cannot be volitionally executed, not from a loss of the representations of the movements concerned, but from inhibition of them. Strümpell holds that the

transmission of the wish for a particular action to the central somatic motor (cortical motor) centres is interfered with.

(2) To suggestion, which, as Charcot has shown, may be (*a*) hypnotic; (*b*) traumatic; and, according to the author, may originate (*c*) through the intermediary of sensations of numbness, heaviness, or deadness in the limbs, which are set up in nervous persons by emotional (pyschical) disturbances, and thus bring about the idea of loss of power.

(3) To auto-suggestion (Möbius and others). Such an idiogenetic origin of hysterical paralysis cannot be gainsaid in certain cases, but is not true for all.

(4) The origin of paralysis as a sequel to convulsions is not always due to exhaustion of cortical motor centres, for a fresh convulsion may remove the paralysis, but may be due to inhibition of them.

(5) Lastly, as atrophic paralysis (chronic anterior poliomyelitis) may follow muscular over-exertion, it is reasonable to suppose that in hysterically-disposed persons a condition of functional exhaustion may be brought about in a similar way.

The kind of paralysis which results from special disturbance, or dissociation of the different acts which are accomplished by one group of muscles, whether these movements are simple or reflex, is typically exemplified in the affection which Charcot and his pupils have called astasia-abasia, but as this affection is only one of many, some general term for this form of paralysis is needed. M. Babinski,[35] thinks the term "systematic paralysis of the pyscho-motor functions" or more briefly, as these paralyses are generally, if not always, hysterical, "systematic hysterical paralysis," using systematic in the sense of belonging to a system, would be a suitable one. We fear, however, that this term will not meet with favour, as it seems to us to lack precision, and to be liable to cause confusion, as the term "system" diseases of the cord is well known in quite another (anatomo-pathological) sense.

M. Babinski's first observation is that of a woman of 50, who contrary to what is observed in astasia-abasia, could walk and stand upright, but could not voluntarily perform simple movements of flexion or extension of the toes, feet, or legs, when lying down.

M. Ballet[36] lately published a case in which the patient was affected with hysterical facial paralysis which was only shown in the movements necessary for speaking. M. Parinaud has shown in hysterical ophthalmoplegia that voluntary movements of the ocular muscles are abolished, whilst the reflex or unconscious are preserved.

M. Babinski reports a case in which when the patient spoke or whistled both sides of the face acted well and equally; the left side of the face also acted well alone, but movements of the right upper lip and right side of the labial commissure were almost impossible. He does not give the case in detail, but we suppose that a limited nuclear lesion was excluded. Another patient suffered, after slight loss of consciousness, from paralysis of the right upper extremity, together with partial loss of sensibility and affection of special senses on the right side. The right side of the buccal commissure lay lower than the left, and the right naso labial furrow was partly obliterated. In any movement of the face, however, the muscles of the right side of the mouth acted well, those of the left side very feebly; this applied both to bi- and unilateral movements. The tongue was put out to the right, but could be moved freely to the left or in any other direction, so that the symptoms were paradoxical. In a third case of a man affected with left hemiplegia, hemianæsthesia and special sense affection, unilateral movements of the face were almost completely abolished, but in the act of whistling and blowing, the muscles of the left side, without executing the movements appropriate to the action, entered actively into contraction, behaving quite differently from a case of organic disease. [This case does not seem to us to be absolutely certainly one of hysteria; the paralysis came on after loss of consciousness lasting six hours, and the patient was a man of 56 years of age.]

The author thinks that in cases of hysterial facial paralysis there is almost always some singularity, foreign to the symptoms in cases of organic disease, which indicates the hysterical nature of the affection, and that the deviation of the mouth is probably due to a contraction of the muscles on the side on which the commissure is highest, and not to a paralysis of the muscles on the apparently paralysed side; that they are cases of spasm, in fact, and not of true paralysis.

Dr. Knapp[37] reports a case of paralysis agitans without tremor, showing the phenomena of propulsion and retropulsion, associated with attacks of inability to walk and with the preservation of muscular force and co-ordination with ability during the attacks to make various co-ordinated movements akin to walking. The author points out that there is not sufficient evidence for the French view that astasia-abasia is a symptom of hysteria. In the course of education, functions such as walking have been assigned to cells at a lower level than those of the cortex, below the level of consciousness. The process of walking is differentiated from other movements of the legs, as writing from those of the hands. In pure cases of astasia-abasia the motor apparatus is at least to a certain extent in working order. Hammond suggests in these cases loss of power of muscular adjustment, but there is no evidence of disorder of the muscular sense. It is only or chiefly when the action of the highest centres is taken off and the subconscious centres are called into play that abasia becomes manifest. Charcot regards these centres as spinal; Salemi-Pace and Hughes further attribute the trouble to failure of memory in the spinal cells, a partial spinal amnesia. The author finds difficulty in admitting the spinal cord origin of the symptoms, and thinks that the affected mechanism is higher up, and that "we can more easily imagine in the brain, below the highest layer of motor cells or those that act only by conscious volition, a subordinate group of cells in the layer of the subconscious . . . . which has been educated to preside over the movements of walking." A breaking up of the associations between the cells would result in a retention of voluntary movement in the legs, but a loss of the automatic power of walking. He thinks that the results of hypnotism show that "in the field of the unconscious ideas of inability to walk have developed, and by a process of suggestion have acted on the lower cerebral centres for automatic walking." This would bring astasia-abasia under the heading of the association neuroses.

The process is distinct from that of agoraphobia because in the latter some external factor presents itself to consciousness, the attention to it is morbidly increased, and the idea thus aroused dominates the field of consciousness and inhibits voluntary effort. In true insanity of doubt, which is again different from agoraphobia and astasia-abasia, the imperative representation is ever present in consciousness, dominates the entire life and is independent of the patient's surroundings.

In a certain small percentage of cases of astasia-abasia the affection is not an association neurosis, but allied to the intention psychoses—agoraphobia, claustrophobia, &c.

The commonest form is the paralytic; in others the motor disturbance takes the form of spasm or ataxia.

The prognosis is good, especially in children; adults often relapse. The author classifies the varieties in two ways:—

$$\text{Astasia-abasia (1)}\left\{\begin{array}{l}\text{Paroxysmal}\\ \text{Continuous}\end{array}\right.\text{(2)}\left\{\begin{array}{l}\text{With distress (intention psychosis.)}\\ \text{Without distress (association neurosis.)}\end{array}\right.$$

So far as the writer's experience of astasia-abasia goes, he has found the common stigmata of hysteria present in adults absent in children. As regards the nature of the affection, the brain probably acts on the muscles through groups of cells in the cord arranged to form a mechanism for each distinct movement such as walking, the cells discharging in definite order, sequence and time, and so regulating the force, duration and onset of the contraction of every muscle or part of a muscle concerned in the various parts of the movement. Disorder of these cord mechanisms would exactly produce the symptoms of astasia-abasia. We have direct evidence of these centres in the cord, but the existence of "subconscious cerebral centres," distal to or on the efferent side of the cortical motor centres, which the author supposes, is entirely hypothetical.

Möbius,[28] in criticising Blocq's observations on astasia-abasia, observes that he would have done better to have prefixed the adjective hysterical to the name, thus indicating the pathogeny of the malady, and that it is a symptom and not a disease. He points out that astasia-abasia is practically not met with apart from hysteria. He emphasises the fact that the touchstone for all hysterical symptoms is that the patient is unaware of the psychical origin of his troubles. As regards the patient's consciousness, an hysterical is exactly like an organic paralysis; he knows as little of the origin of the one as of the other, and does not distinguish between them. The patient sees that his legs do not bear him, but has no further notion why that is so. This is the reason why hysteria is assigned to neuropathology and not to the psychoses (psychiatry). Scientific researches first disclosed the connection of the hysterical symptoms with (antecedent) psychical processes.

After criticism of Binswanger, Ladame and Blocq's observations, Mobius goes on to say:—

In hysterical loss of power, the idea of the loss of power is the cause of the paralysis. Whether this idea starts up suddenly (as in an accident), or is gradually developed (long confinement to bed), or arises during a dream or in the waking state is of no moment. Its operation takes place on the basis of the hysterical disposition in the one case, and in the other on a special mental state existing at the moment. Of this disposition of mind we have no clear idea. It must be similar to the hypnotic state, and there must be a certain emptiness of consciousness, in which, so to speak, the throne is for the first comer. We know that, besides hypnosis, states of terror and depressing influences (insomnia, hunger) can produce such a condition. If this be granted, we have yet no conception of the manner in which the idea, whatever diseased condition it produces, exactly works, but have to content ourselves with analogies, which we understand as little, to the paralysing influence of terror and the like. But the clinical touchstone of hysterical paralysis is that the antecedent causal psychical processes do not enter into consciousness, the patient stands as much outside the paralysis as he does outside one of organic origin, and even the physician, if he is not instructed in its forms and tendency to disappear under psychical influences, may mistake hysterical paralysis for organic disease. Further, it is characteristic for hysterical disorders to affect the young, not seldom children, and to be associated with the so-called hysterical stigmata which indeed cannot be directly caused by idea, but which may be considered to be dependent upon idea in the same way that in the healthy bodily disturbances are dependent upon the affections (emotions).

In their remarks on the first of two cases of male hysteria, Drs. Bourneville and P. Sollier[29] note (1) alcoholism in the father and grandfather and various nervous disorders in the mother, brothers, and sisters; (2) development of hystero-epilepsy in a patient who was late in learning to walk and talk, suffered from nocturnal incontinence of urine, mental irritability and instability, tendency to violent actions, &c.; (3) the patient was a Jew. "During its whole existence, up to the time of the French Revolution, and even now in some countries, this race has for centuries been especially exposed to a number of strong emotional excitants, which have endowed not only individuals but the race itself with an ethnical nervous state, rendering its members particularly predisposed to all nervous maladies." In support of this opinion there, the following local statistics for the Department of the Seine are adduced. In 1875, at the Bicêtre Asylum there were fourteen Jews out of 538 insane patients. If the proportion at the other asylums was the same, that would give 188 Jews out of 7,248 patients. The total population of the Department is 2,220,060 persons, of whom 24,319 were Jews. The proportion of insane was therefore, in the case of Jews, seven per 1,000, whilst for the rest of the population it was only 3·2 per 1,000. (4) When the convulsive attacks came in succession during a rather long period, the temperature rose a little above 38°, but never attained the height it reaches in the status epilepticus.

Professor Popoff[30] records the case of a patient, a peasant of twenty-one, suffering from regular, rhythmical tremor of small amplitude, increased by observation, by attempts to walk or to make any voluntary movement. The tremor could not be voluntarily controlled, and was increased markedly if efforts by the attendants were made to stop it. There was loss of power and sensation in the limbs, and the fields of vision were contracted. Mechanical irritability of the muscles and the tendon reflexes were increased. The unusual feature of the case was that since the age of eight the patient had been liable to attacks of trembling in the legs, with loss of sensation over them. For some days after the attacks, a feeling of fatigue prevented the patient from working. The attacks came on at intervals of one month, owned no apparent cause, but could be provoked by emotion, *e.g.*, a fright, and lasted two or three days. They ended quite suddenly.

Professor Popoff satisfied himself from personal observation, that the attacks had precisely the above characters, and points out the singularity of their periodicity and of the absence of any exciting cause for them; he can find no exactly similar case on record, but one reported by Chambard (*Hemichorée et tremblement hystérique. L'Encephale*, 1881), most nearly resembles it, as tremor was the most prominent symptom of hysteria present, and came on in attacks, but these had no regular periodicity, and were mixed with hysterical seizures of another kind.

Dr. Kinnosuke Mirna[81] has collected twenty-eight cases of flaccid hysterical monoplegia from medical literature between the years 1885 and 1890, to which he adds three cases of his own. Twenty-three were men and eight women; for the most part the patients were engaged in laborious occupations, involving much muscular effort; six of them were exposed to the risk of lead poisoning. In thirteen nervous inheritance was clear, in eight doubtful, in nine the family history was unknown. During childhood the majority had suffered from incontinence of urine, moral perversions, convulsions, &c. Three patients were alcoholic, there was a relapse of the paralysis in four, and in several hysterical convulsions.

An injury was the most common exciting cause. In six cases there was plumbism; in three a moral emotion preceded the development of the paralysis; two patients were attacked after sleeping in cold and damp; and in two the paralysis appeared after fixation of the limb in a fracture apparatus.

Paralysis came on either immediately after the injury, or after an interval of time, varying from one to twelve days.

Affections of Sensation.—Hemianæsthesia in nine cases; in fifteen the anæthesia was limited at the root of the limb affected by a line drawn perpendicularly to its long axis, but in four others (brachial monoplegias) it affected also the same side of the head and neck and the upper part of the body as far downwards as the epigastrium or umbilicus. In one case (of the author's) there was no anæsthesia present.

Trophic disorders were absent in eighteen, present in nine, consisting of atrophy of the paralysed muscles, and in two also of the bones; in one there was an elevation, in two lowering of the temperature of the paralysed part.

Tendon reflexes were exalted in nine, diminished or abolished in nine, normal in three, and in fourteen cases not recorded. Special senses: in nineteen cases contraction of the visual field, in seven monocular polyopia, in four diminution of acuteness of vision, in two dyschromatopsia; enfeeblement or loss of taste in eighteen, of smell in seventeen, of hearing in twelve. In two patients special senses unaffected; in three not recorded.

Other signs of hysteria noted are, ten times anæsthesia of the pharynx, nine times tender or hysterogenic spots, seven hysterical attacks, and four times "ovarian" tenderness. In twenty-two cases the condition yielded to various methods of treatment; in four it was very obstinate, persisting in spite of all efforts, and in five there were relapses.

Under the title of *Akinesia algera* Mobius[m] describes an affection which consists of loss of voluntary movement on account of pain on movement, without there being any tangible ground for the pains. It appears in people of neuropathic tendencies, family and personal. After over-excitement, a condition of nervous weakness develops. Pain *first* affects only the larger, afterwards nearly all movements. In part it accompanies, in part follows, movements and not only in the moved, but also in other parts of the body. At last there comes complete loss of power, in which the parts appear paralysed and helpless. In addition, the signs of neurasthenia appear: disturbed sleep, weak voice, incapacity for mental exertion, pains and pressure in the head, painful sensations in the back. Undoubted hysterical symptoms were not present in the patients observed. As to the issue that is not as yet determined. We must admit that recovery is possible, yet the history of the two patients proves that mental disease may be added to akinesia algera. The author hesitates to draw general conclusions from only two cases.

I.—The first patient, age 33, was a school teacher. The mother and her family were healthy. The father suffered from paranoia, hallucinations and delusions of persecution, and lost his employment. The family life was much prejudiced by want and the (mad) humours of the head of the house. The patient had thus a melancholy bringing up, and was often terrified by his father. He did very well both at school and university, worked immoderately, and gave great promise for his future career. On becoming a teacher he overworked himself, and was esteemed by the authorities and beloved by his pupils as an ardent, careful teacher. Soon after obtaining his post he married. In 1887 he got a pain in his head and suffered from sleeplessness. After a holiday he was, for a short time, better. In 1888 the head pressure returned, and mental work became impossible. He could still conduct a conversation and write a letter, but all heavy work and original or consecutive thought was impossible. "The head simply forbade it." In spite of several trials of different methods of treatment his condition grew worse; after coming out of an institution for nervous disorders he passed for three weeks into a peculiar condition of which he had no recollection afterwards, and in which he took no notice of his surroundings, except occasionally to bewail his fate. Treatment by prolonged warm baths somewhat relieved his symptoms. In the spring of 1889 he was ordered to take short walks in the garden, and treated by overfeeding and rest; this resulted in improvement, but he had to break off the treatment and return to a university institution. Here he was ordered to carry out movements with his limbs, and this made his condition worse, he suffered pains in the back, "muscle pains," and "nerve pains." His arms were now affected, and small movements of the hands gave rise to long-lasting pain. A fresh course of treatment by rest gave no better result, and in September, 1889, the author first saw him.

The patient lay on his back. The legs could be moved in every direction, but the movements were feeble and of small extent; no resistance to passive movements. The arms and fingers were in position of extension; movements of both the patient only accomplished to a small extent. Passive movements of the hand and fingers met with a dead resistance, but the patient would not permit much display of force. The patient refused to kneel or to get up. Movements of the head were free and power good; he moved his head without hesitation, and did not complain of fatigue of the neck muscles.

The nutrition of the muscles was in general not bad, but there was slight though evident atrophy of the interossei in the left hand. Skin reflexes normal. Tendon reflexes normal in the arms, very brisk in the legs. Ankle clonus distinct in the left, faint on the right side. No diminution of sensibility anywhere, on the hands and forearms was some hyperæsthesia, light touches being felt as painful. The skin was normal except over the fingers, where was a high degree of "glossy skin." There were no disturbances of the special senses of any kind. The mental condition was unaffected; the intellect clear and good; he could only read a short time, or dictate a short letter; if he tried to do more pains in the head came on and rendered all thought impossible. K. felt that he must avoid all movement on account of the pain excited. The pains were directly related to the amount of movement, were worse in the arms, and, at the same time, he was not quite free from pain when completely at rest. Some days he was much worse than others. Appetite and sleep were good; the bowels acted regularly, and sexual desire was not entirely lost. Lungs and heart normal.

Passive movements systematically carried out made him so much worse that they had to be desisted from. Various drugs were tried, including two thorough trials with opium, without any effect on the condition. Attempts at hypnosis failed. After some months there was some improvement: the pains in the head disappeared, and he was able to read for long periods. He regained some power of movement in the legs, the tendon reflexes were less exaggerated, but the hands did not improve. In 1890 he had an attack of influenza, which did not affect his general condition. After this he showed very gradual improvement, until at last he was able to move himself from his bed on to a sofa placed alongside it. During the author's absence on a holiday, his *locum tenens* said that possibly K. was a simulator. This deeply grieved him, and he went back, but again very slowly made some degree of recovery. In January, 1891, the condition is that he lies on his back, can move the legs freely in bed but cannot stand a walk; he does not use the hands, but can carry out small movements of the fingers without pain; he has to be fed and uses the bed pan; no muscular wasting nor increase of tendon reflexes, the mental condition is normal, except for a certain irritability and readiness of fatigue.

II.—Miss L., a music teacher, 43 years old. Her mother's family was nervous and the mother herself was paralysed during the last years of her life. Up to 20 years of age L. was healthy, but eccentric. She had attacks of "trembling spasms" after the death of her parents. She was studying music very hard in her 20th year when the pains in her hands began, and grew worse, so that she could not use them; there was also contraction of the hands, so that she had to keep them in position of flexion. She was incapable of any mental exertion, and slept badly. A year afterwards pains in the legs began. At times the pains were worse, so that the joints could not be moved. The illness lasted ten years. Dr. Bärwinkel, under whose care she was, considered the symptoms to be due to general hyperæsthesia of the joints, accompanied by contractures of the fingers, hyperæsthesia of the lower extremities and vertebral joints; hysterical attacks, lasting some seconds, with loss of consciousness, and clonic spasms of the upper extremities, occurred. Treatment had no influence. She recovered, and for ten years remained well and earned her living by teaching the pianoforte. In the summer, 1889, she strained herself by carrying a handbag on a journey, and also in nursing a sick niece, to which she attributed the fresh attack, which came on with pains in the hands and back, and excitability and weariness. By the middle of October she was already bed-ridden; Dr. Mobius first saw her in November, 1889. The fingers and hands were kept in semi-flexion; as soon as the flexion at the wrist-joints was gently overcome they opened themselves automatically. Passive extension met with no noteworthy opposition. Movements of the hands and arms could be carried out, but with pain, which lasted for some time afterwards in hands and elbow-joints. The legs were freely movable. She could stand and walk from one room into another. This was the extent of her powers, any further efforts giving her pain. There was no disturbance of the special senses, of common sensibility, or of the superficial or deep reflexes. No muscular wasting. Appetite good. Bowels regular. Sleep bad. Catamenia regular. The mental state showed no signs of disturbance. The patient's friends described her as an intelligent, somewhat cold, very strong-willed person. In January, 1890, she had the influenza, which almost seemed to have a good influence over her condition; but some weeks later a bad attack of migraine left her worse than before. Attempts at hypnosis completely failed. Then came a period of improvement, in which the patient was able to stand a little, to sit up, and to read. She went into the hospital, and there passive movements were carried out which resulted, for a time, in improvement; but she relapsed after a short time, and attributed this relapse to being obliged to carry out movements in spite of the pain caused thereby. She made two attempts at suicide, lay motionless on account of the pains that arose from every movement, could no longer sleep, and had lost all hope. Subsequently she became demented, heard voices, had delusions, and thought her food was poisoned.

The two cases correspond very closely in the chief points. Both patients had a bad inheritance; both were, before the illness, already "dégénérés," and suffered from mental over-excitement. The symptoms came on gradually, together with those of neurasthenia. In K. the latter were well-marked, and in L. there were also hysterical symptoms. A slight difference between the two lies in the fact that L.'s first illness began with a contraction of the hands, and not with loss of voluntary movement; but of the second illness, which alone was observed by the author himself, there was no sign of contraction, the symptoms being similar to K.'s. Further, in K.'s case there was exaggeration of the deep reflexes and wasting of the muscles of the left hand. The last might be attributed to the compression of the bandage, or be dependent on the disease. The latter appears to me to be the more probable, and both symptoms might be due to hysteria, for a like degree of excessive myotatic irritability and muscular wasting has been shown by Charcot and his pupils to occur in hysteria. In both patients the pains were very obstinate, and all attempts at compulsion and the influence of emotions made them worse. The first case has not yet ended, and insanity has brought the second to a provisional ending. The question first of all arises, what is the nature of the morbid process? The inherited neuropathic taint, the presence of nervous hypererethism, of neurasthenic and hysterical symptoms, the peculiar course and the general features of the disease lead to the conclusion that it is a psychosis. If we attribute akinesia algera to paranoia in the widest sense of the word, a further definition of the variety is also desirable. The conception of neurasthenia would have to be extended to include akinesia algera; and one can, on the other hand, adduce grounds for the hysterical as well as for the hypochondriacal nature of the symptoms. All depends on what we mean by these terms. Briefly one may define as hypochondriacal, those bodily disturbances of whose psychical mechanism the patient is conscious; and as hysterical, those in which the connection between perception and material disturbance does not enter into consciousness. In this sense the pains of akinesia algera are hysterical. Should exception be taken to consideration of the pains as material symptoms, the reply is that deficient sensation and hyperæsthesia, though really subjective, are commonly reckoned as material symptoms. It might be further objected that the estimation of a symptom as hysterical does not depend on theoretical speculations, but on the presence of other undoubted signs of hysteria. This view, however, is contradicted by the existence of many forms of monosymptomatic hysteria. Often the diagnosis of hysteria can only be made by exclusion.

Although Möbius regards the pains of akinesia algera as hysterical, he cannot consider the disease as simple hysteria. There is an analogy between cases of analgesia algera and those of general anæsthesia, which have lately been published by Kinkenberg, Heyne, and von Ziemssen. Hysterical manifestations in analgesia algera are combined with hypochondriacal and neurasthenic symptoms, and taken as a whole the disease resembles more closely severe hypochondriasis than simple hysteria. W. Neftel[83] is quoted by the author as having described a condition very like that of analgesia algera under the name of atremia. In these cases (four in number) the patient had always a nervous heritage, and fell ill through over-excitement; there were photophobia, incapacity for mental or corporeal exertion, because both evoked general indisposition, paræsthesia, diarrhœa, and loss of sleep. The patients lay continually in bed, and all attempts to make them walk aggravated the symptoms. Treatment was without effect. Neftel considered atremia as hypochondriacal insanity. The chief difference between atremia and analgesia algera is that in the former, walking, standing, and sitting are impossible, whilst the limbs can be freely moved in bed without complaint, whilst in the latter all movements of the limbs are entirely desisted from. Atremia bears the same relation to analgesia algera as abasia-astasia to hysterical paraplegia. In atremia walking produces only disturbances in the general condition, and paræsthesia in the head and back, whilst in analgesia algera the pains are in the parts moved. In other respects the two conditions correspond. Binswanger describes a similar condition of loss of voluntary movement on account of pain, and regards it as an episode in the the course of hysteria. As already mentioned, so far as the above observations go, treatment has proved ineffectual and the prognosis is bad.

Erb[34] thinks that it is still doubtful whether the symptom-complex so clearly painted by Möbius can be regarded as a distinct morbid entity; time and further observations will show, but there is no doubt as to the practical utility for the advance of knowledge in thus circumscribing a group of symptoms. As Möbius points out, the affection on the one hand approaches the psychoses, and on the other is closely related to hysteria, and especially to neurasthenia. As for the cases that Erb can recall to memory, he has referred them partly to the severe forms of neurasthenia, which especially in effeminate persons present the highest grade of irritable weakness and exhaustion of the nervous system, partly to that affection which we call spinal irritation. They either affect undoubted hysterics, or they appear under the form of hypochondriacal psychosis.

He considers that there are essential points of difference between spinal irritation and akinesia algera. Erb has seen several similar cases, and come to the conclusion that it is very difficult to treat them successfully; he reports the following case as illustrating in a striking way the symptoms of akinesia algera.

The patient was an Englishman, aged 47 years, in the diplomatic service in Italy. He came of a highly neuropathic but talented family. The patient was always weak and nervous, and overworked himself at school and college. He had been ill since his twenty-second year; the illness began, after excessive mental overwork, with inability for gymnastic exercises. Later, exercise of any kind caused pains in the heart, palpitation, and various vaso-motor disturbances. In January, 1873, every slight movement brought on pains in the heart and palpitation; he was treated with injections of strychnine, which had an untoward effect, so that he could neither stand nor sit, but took to bed. After fourteen days he was able to stand for a short time, and to sit up longer, and passed six months partly lying, partly sitting. The head was free from pain, and mental work could be well carried out. Attempts to rouse him and to make him exert himself only ended in aggravation of his condition. In the year 1873 he lost the power of reading and of writing, on account of the pains they excited; later he could not even bear reading aloud, though he could still sustain a conversation. He was obliged to keep constantly lying down.

From 1878 to 1888, the patient remained in the same state: he lay in a horizontal position day and night. All the functions of the body were normally performed, except that sleep was bad, and sexual power abolished. From 1888 the patient made again attempts to leave the horizontal position, but only succeeded in standing for the space of four minutes; he could not bear anyone to talk to him, nor for more than two or three words to be said to him, although he could talk freely enough himself. Two new attempts to get him out of the horizontal position failed; as soon as he was raised a few inches the pains came on again. Although he had thus passed fourteen years on his back, he had not lost his sense of humour, and had even made himself some reputation as an author. The patient struck Professor Erb as a highly gifted, intellectual, and amiable man. He could not bear Professor Erb to speak any sentence longer than two or three words, but did not seem to remark that he could quite well bear his own long-flowing speeches. There were no special nervous or hysterical symptoms. There were no signs of organic nervous disease. All forms of treatment had been tried, without any success.

The above case speaks for itself. In the leading features, in the strong neuropathic inheritance it falls under Möbius' definition, and it may be emphasized that in this case symptoms of neurasthenia, hysteria or of any psychosis were absent. Whether the patient from psychical influence or gradual disappearance of his notable hyperæsthesia will ever recover from his lamentable condition, remains undecided.

(*To be continued.*)

BRAIN.

PART II., 1894.

Original Articles and Clinical Cases.

IMPERATIVE IDEAS.[1]

BY D. HACK TUKE, M.D., F.R.C.P., LL.D.

I CANNOT do better than take for my text these words of a distinguished member of this Society:—" The slightest departures from the standard of mental health are to be studied, and not only the cases of patients who require to be kept in asylums. Indeed, the slightest cases are the more important in a scientific investigation."—(Hughlings Jackson.)

In speaking of imperative ideas I refer to those cases in which a person would not be regarded as insane, although the mental trouble may be as distressing as it is in actual insanity, and may at last become so pronounced as to render an asylum a relief to the patient himself. In our official classification of mental disorders we have not been in the habit of very clearly recognising, as a separate group, even this advanced stage, partly, no doubt, because if the patient becomes depressed, as frequently happens eventually, the case is classified under melancholia, or if he comes to interpret his imperative ideas as satanic or divine, his case is relegated to the head of "delusional insanity." In many instances such cases are diagnosed as *neurasthenia*, and there is no doubt that in some patients there is nervous exhaustion, but I think that the characteristic feature of the malady under which these persons suffer is too much lost sight of by so general and vague a term.

---

[1] Read before the Neurological Society, March 1, 1894.

Symptoms.—With regard to the symptoms, they assume different forms. With some, the torment is that certain ideas or words arise with painful frequency and vividness. The thoughts which dominate the mind with morbid persistency are generally of an unusual and unwelcome character, and the words which the individual is strongly impelled to utter, are altogether contrary to his habit or desire, and if uttered, this is in spite of his strongly exercised will. Then there are persons who invariably touch some object in passing it in the course of an accustomed walk (*Délire du toucher*), the antithesis of which is seen in the dread of touching certain objects at all, a true mysophobia. Then there is a form of imperative ideas which Charcot and Magnan have termed onomatomania. These physicians have comprised under it a group of symptoms in which a word or a name plays the chief part, and have maintained that these mental troubles arise from what they call a loss of mental equilibrium. They also held that this condition is always associated with an hereditary taint of insanity, which I think is going too far. They included under this term, the agonising search for a particular name or word, a more or less irresistible impulse to repeat it again and again when found, or to attach a disastrous signification to certain words. (*Archives de Neurologie*, Sept., 1885, p. 137.)

One subdivision of imperative ideas, and a very striking one, is the insanity of doubt (*Maladie du doute*), and termed by the Germans Zweifelsucht and also Grübelsucht, or as we should say, beating one's brains about some question. Closely allied is the insanity of the metaphysicians—Schöpfungsfrage. According to the late Professor Ball, of Paris, the malady of the metaphysicians constitutes a subdivision of the malady of doubt. Then we have arithmomania, or the morbid desire to count without rhyme or reason, or to make interminable calculations; to reckon up the number of books on the table, or the buttons on a waistcoat, or the same letters occurring in words. Almost endless, however, are the terms which have been introduced to label the particular form which imperative ideas assume. Certain alienists simply revel in coining terms for every imperative idea which they have ever met with. They give us not only agoraphobia and claustrophobia, acrophobia (the dread of being in high places), coprolalia (the involuntary use of obscene words), but astraphobia (the causeless fear of an approaching thunderstorm), zoöphobia (the fear of certain animals), oikophobia (the morbid fear of home), and to crown the complexity, bélonéphobia (the morbid fear of pins).

However allowable it may be for the sake of convenience thus to multiply terms, they must not distract our attention from the fundamental characteristics common to all imperative ideas. These are, their automatism, the overwhelming and recurring tendency to be haunted by a certain idea, to perform certain acts, or to use certain expressions against the will, *without delusion* (at least in the first instance), and indeed with a consciousness of the utter uselessness and absurdity of dwelling upon these ideas, and of the things which are done or said. Momentary relief may follow the discharge, but ultimately mental distress. The imperative conception may occur spontaneously, free from any external stimulus, and apparently (though sometimes only apparently) unconnected with antecedent thoughts.

It must also be remembered that although we speak of imperative ideas, some cases would be more correctly termed *imperative emotions*, and others *imperative impulses*. But that which I have more especially in view might be misunderstood if I used the latter terms, although the sufferer from the ideas in question suffers mental anguish in consequence, and often has irresistible impulses.

I am anxious to bring into prominent relief the absolute innocuousness and sanity of a large number of imperative ideas and acts. Take a few illustrations drawn from persons among my acquaintance, some of them being well-known physicians.

(1) A lady frequently wakes in the night with a sense of impending danger, conscious that there is no ground whatever for it. She obtains relief by giving three raps three times on the bedstead.

(2) A gentlemen, in attempting to open a door which he has no reason to suppose is stiff, for example in his own room, employs force to do so, and strikes the architrave in order to assist his efforts to ease the door. It has become a habit which he is unable to resist. I am not aware of any explanation.

(3) A medical friend has always an apprehension of something dreadful about to happen upon a door being closed, although not believing for a moment that anything will happen. Here again, no explanation can be found in any circumstance which previously happened calculated to associate the occurrence with an accident.

(4) Dr. ——— has the electric light in his house, and puts it out before retiring to bed. After going upstairs into his bedroom, he has troublesome doubts as to whether he has really done so. He returns in order to be quite certain, although he was certain when downstairs a few minutes before. Similar experiences are common, and may be explained by having performed an act so automatically that it had not been consciously registered; for, certainly, when a person deliberately and consciously performs an act, it could normally be recalled within a short period of time.

(5) Dr. ———, when on a height, experiences a strong and almost imperative tendency to throw himself over the edge. This does not arise from vertigo, nervousness or the slightest temptation to end his days. It is a blind impulse which he knows to be totally unreasonable, but which he finds it difficult to resist.

(6) Dr. ——— has, or had, an imperative idea, the exact opposite to Dr. Johnson's. He feels that he must avoid treading on the divisions between the flags of the pavement, or that something would happen of a serious nature. He is well aware of the absurdity of the idea.

The cases which I will now describe are, with some exceptions, illustrations of a graver mental condition, although the symptoms in the first instance, at least, are consistent with sanity, the subject of them being quite able to perceive their groundlessness and their ridiculous character.

(7) I had under observation a gentleman who was the last man in the world to use profane language, but who had the very greatest difficulty in preventing himself doing so in church and sometimes

while walking in the street without the slightest cause of irritation. His wife was surprised to see him give a sudden jerk from time to time, and was not aware of the cause. He, however, told me that these spasmodic movements arose from his attempt to rid himself of his trouble and avoid using bad language, and as a matter of fact the incipient motor act was checked at that stage. For this painful condition one has for long been accustomed to employ more especially the term *obsession*, as indeed, the French do for all imperative ideas. There is, however, the objection to the word, in relation to a patient, that it encourages the idea, only too common, that some demoniacal agency is the cause of the trouble. In fact, in the case now spoken of, the gentleman was disposed to regard his trouble in this light, and was partially relieved when informed that it was nothing of the kind. I will only add that he was ultimately greatly benefited by a sharp attack of gout.

(8) Next, as regards the tendency to touch objects, there is the well-known instance of Dr. Johnson, whose mental peculiarity is thus described by Boswell :—" At that time the broad flagging at each side of the streets was not universally adopted (in London) and stone posts were in fashion to prevent the annoyance of carriages. Upon every post as he passed along he deliberately laid his hand; but missing one of them when he had got to some distance, he seemed suddenly to recollect himself, and immediately returning back, carefully performed the accustomed ceremony, and resumed his former course, not omitting one, till he gained the crossing" (" Life," vol. i., p. 497). Dr. Johnson also illustrated the counting mania, and indeed I am not sure that the touching of the posts in the streets was not also due to this arithmetical passion. We are told of his anxious care to go out or in at a door or passage, by a certain number of steps from a certain point, or at least so that his left foot should constantly make the first actual movement when he came close to the door or passage. Boswell adds:—" Upon innumerable occasions I have observed him suddenly stop, and then seem to count his steps with a deep earnestness; and when he had neglected, or gone wrong, in this sort of magical movement, I have seen him go back again, put himself in a proper posture to begin the ceremony, and having gone through it, break from his abstraction, walk briskly on, and join his companion."

Johnson, it should be added, laboured under an hereditary taint of insanity, and had time permitted I could have given an instructive account of his mental history.

(9) In the following case, that of a lady, sent to me by Dr. Strangman Grubb, of Ealing, the most prominent and distressing symptom was the incessant tendency to count a certain number of times before performing the simplest act. It can readily be imagined what mental fatigue this imperious necessity occasioned. To be haunted by what the late Professor Ball called an "intellectual impulse" would be bad enough for a few days, but to be the sport of this tyrannical suggestion for years (in fact she had been six when I was consulted about her), could be no other than an intolerable burden. She described to me how even in the night, she could not turn over in bed (unless she did it with great rapidity), nor remove the watch from under her pillow, without counting. In the morning she generally experienced the greatest difficulty in getting out of bed without being obliged to go through the same numerical process. Her friends would hear her step on the staircase on coming down to breakfast, and then several small raps were made by her feet, whilst she was prevented proceeding further in consequence of having to count up to a certain number before descending another step. She says that if anyone had been behind her and said "Come along, you go downstairs," she would have been able to do so, without having to pass through this tedious process. Imagine her now at the breakfast table, wishing to take up the teapot; a considerable time would elapse before she was able to seize the handle, the only means of accomplishing this feat being the same wearisome process of counting, generally to ten or multiples of ten. Suppose now she took a walk, she could not open the front door without counting, and having passed into the street she would probably be impelled to return simply to touch the door handle. She had no pleasure in looking at the shop windows or at pictures, for she had to count a certain number of times before her enemy allowed her to regard them. She was frequently compelled to count the number of times she breathed, and she could not avoid counting her steps as she walked along the road. Again, in saying her prayers, she had not proceeded far, before she was under the dire necessity of returning to the first word and going through the prayer again. At church her difficulties were great, as may be supposed, and to take the prayer book into her hand was an exhausting operation from the act of counting which it involved. Sometimes she would put the book down again, and take it up in a different way. To open a drawer was a very serious matter indeed. Before blowing out a candle she had to count so many times, and when using her needle, she put it into

her work as often as twenty times before making one stitch. The malady of doubt entered largely into this state of mind. When reading, she often worried over the doubt as to the paragraph in the page (or if there were two columns, which of them) she should read first.

When asked whether she had ever thought there was poison in her food she said not, but she answered rather doubtfully. On the following day I received a letter from her to say, that although she had never thought that her food had been purposely poisoned, she had sometimes a nervous fear that something *might* have accidentally got into it, adding that she had sometimes refrained from eating things from just the same kind of feeling which had on many occasions prevented her saying the things she wished to say. She remarked in regard to both the feeling of hesitation and the habit of counting, that "when very bad it produces a feeling of mental tension, which is very trying, not to say painful, and which makes it extremely difficult to occupy myself with any ease or pleasure. One feeling which has made me afraid of breaking through this habit of counting has been a fear lest the thought—sometimes one and sometimes another—which has connected itself with the counting might fix itself in my mind and be worse than the counting."

About nine years ago, when in church, something was wrong with the heating apparatus, hot air escaped, and she felt as if she would be suffocated if she remained. Everything became dark and indistinct to her as she went out. In consequence of this she was depressed for a week, and some of her subsequent troubles followed upon it. The dread from which she had never previously suffered, of being buried alive arose; and she was afraid of walking in her sleep into some churchyard, and being supposed to be dead, when only in a state of somnambulism. She pictured to herself all the horrors of a conscious interment.

Such is a rapid description of the trial under which this unfortunate lady laboured for many years. Perfectly conscious of this strange mental condition, and very bright and intelligent, she was able to discourse upon it, and to suggest what she thought was the process of thought by which she came to preface almost every act of her life by counting. She compared it to a boy running a race, but unable to start until someone calls out "One, two, three and away!" She regards this as the immediate origin of her habit, and that she did in the first instance, and does perhaps even now count, with this object.

Although there is no insane inheritance known, it is of interest to note that one of her brothers died an epileptic, and one of her sisters, who is living, has epileptic fits. A law student, I shall shortly mention, had a family history of epilepsy as well as insanity. In a remarkable article in the *Annales Médico-psychologiques* (Jan. 1890), by Dr. Culerre, the association of onomatomania with epilepsy is insisted upon. In the case I have brought before you there have been no fits of any kind, and she has never fainted, although turning deathly pale when hearing the account of the death of Mr. Bishop, the thought-reader, connected as it was with the suspicion that the autopsy was made when he was not really dead. She does not appear to have had attacks of *petit-mal*, but I elicited the fact that she has at times the feeling of "I wonder am I really doing this?" She has never suffered from vertigo. She has experienced the not uncommon sensation of having been under precisely the same circumstances and in the same place on former occasions. She was a nervous child, but she assures me that she was not naturally undecided, and although not making up her mind very quickly, she had no tendency to vacillation. There has been no hallucination of any of the senses and no delusion. I have recently heard that she has recovered.

The next case is an example of mysophobia and the malady of doubt.

(10) This patient (Watts) came under Dr. de Watteville's care at the West End Hospital for Nervous Diseases, in February, 1893. His attention was not drawn to any mental trouble beyond general nervousness and a feeling of being equal to his work. He regarded the case as one of *neurasthenia*.

Some months afterwards, Dr. de Watteville asked me to see this patient. His mental symptoms were these: He said that his great trouble was that on going to do a thing he always had a sense of doing it wrongly, and after posting a letter or putting something into a drawer, he had an invincible misgiving as to whether it was correctly done. He felt that there was no rational ground for this misgiving. He was also greatly worried as to whether he had taken anything away in his hand from any place which he should not have taken. After getting out of an omnibus he began to think that he had left something behind him; he said that he knew perfectly well that he had not, and yet he could not help having the idea presented to and tormenting his mind. Again he felt from time to time, he knew not why, that he must look up to the ceiling of the room he happened to be in. He had fancies about his clothes being dirty, and often looked at his hands to see whether they were quite clean. He did not complain of mental depression.

On the *physical* side there was slight tremor of the hands. The pupils were dilated, sluggish and equal. His knee jerks were very brisk. With the exception of dreaming his sleep was good. His habits were temperate and he was not a smoker.

Family History.—His mother suffered from "nervousness," and he considered that he inherited it from her. He was the youngest of twelve children, four of whom died of phthisis, and a brother committed suicide. He was not aware of any insane or epileptic ancestor.

Some months later he became worse, and it was quite a relief to him to be allowed to come into Bethlem Hospital as a voluntary boarder.

I will now describe a very striking case illustrating imperative ideas not amounting to delusion in the first instance, but ultimately becoming so.

(11) A. F., aged 58, sent to me by Dr. Wakefield, was able, when I first examined him, to laugh at the notions which arose in his mind, and he would say that of course he knew there was nothing in them. He said that two years previously, the idea came into his mind when he took the Eucharist that blood would come into his mouth. This idea had entirely left him, but only to give place to others. He was a Roman Catholic, and when young he was engaged as an acolite about the altar, and he had, while in the church, strongly impressed upon his mind the pictures and images of the crucifixion. To this he himself attributed a feeling of swelling and pain in the palms of his hands, and his attention was in consequence frequently directed to them. In the night if he awoke he was under the impression that he himself was Christ, but when he got up in the morning he shook off the idea, and was distressed to think that he should have entertained it even for a moment. No stigmata ever made their appearance, but it is not difficult to believe that they might have done so in the course of time, for he got to suspect that blood was flowing from his hands. This led his sister to put out of sight a picture which he had bought representing the crucifixion.

But the most absurd, and to him the most mortifying imperative idea of all, was that he was pregnant. It was not when he first mentioned it to me a delusion, for he could not help laughing when he told me. It originated partly in a dream and partly from reading in a newspaper that a young woman had said that if *she* were *enceinte,* she should drown herself.

Some sensory disturbance was mixed up with his idea, for he said it seemed to him as if his abdomen was of greater size, and yet he added, "I know it isn't." Sometimes he had to resist the idea that flashed into his mind, that he was a woman, and felt that he should like to shut himself up in his room and not see a soul, for very shame. Above all things he was afraid when walking in the street that he should meet a pregnant woman. Well, after a while, he really believed that he was pregnant; he no longer smiled at the idea, but pulled a very long face indeed.

With regard to his *family history,* his mother was subject to mental depression, but not in an asylum. He has a brother and two sisters, and they are mentally healthy. There does not appear to have been any epilepsy in the family.

To complete his history, I should add, that after some time he refused his food, averred there was some obstruction of the bowels, and it became necessary to place him in a private asylum, where he died of pneumonia in about ten days.

(12) *Case of Miss* ——.—This is the case of a young lady who has laboured for some time under the dread of touching anything for fear of infecting others who might touch the same article, and I fear I can hardly maintain that her ideas do not now amount to delusions. The first symptoms arose after influenza. There is at the present time in her mind the fear of infecting herself as well as others, although she does not say that she *knows* it would be so. The result is that the act of washing her hands in the morning is repeated, immediately, again and again, and although she does not own to counting, her friends think that this idea enters into her course of ablutions. She invariably passes the sponge over the upper surface of the arm, one side, then the under surface, and lastly the other side of the limb. She forces herself to lie awake, lest in her sleep she should put forth her hand and therefore contaminate some object near her. What seems a strange inconsistency, is that she is obliged at times to touch objects, feeling in a vague way that if she did not, something dreadful would happen. She has on one occasion burnt her clothes, fearing that they have been infected by her. She says she would have washed them, but knew she would not be allowed to do so.

Then she suffers from the malady of doubt. Endless questions arise in her mind. Which gloves shall I put on? The brown ones? The black ones? and so on. She will spend twenty minutes at her drawer in this state of indecision, conscious that it is foolish to do so. I may here remark that the hopeless indecision under which some sane people labour is akin to, if it does not fall under this form of imperative ideas.

As a fact, this young lady scrubs her hands and arms so fiercely that they are quite rough and discoloured afterwards. She keeps one part of the towel for the face, another for the hand, another for the wrist, and so forth. When I advised her not to think about it, she replied: "But then what would happen, if unconsciously I *did* infect the towel?"

It is important to note that she herself states that this morbid dread began in consequence of having warts on the hands, and hence dreading she would infect other people with them.

While admitting that this case more nearly approaches insanity than sanity, I should not dream of signing a certificate for her.

(13) *Case of Miss B.*—The next case is one of mysophobia of a very similar character to the last. Her imperative ideas have certainly crossed the rubicon between those which are not credited and those which are. I only mention it as illustrating the later stage, although she is not in an asylum. It is interesting as combining the dread of contagion with the onomatomania of Charcot and Magnan. It is important also as showing that a careful analysis of imperative ideas and acts will often explain their origin. Thus while reading a book or newspaper she will suddenly evince signs of irritation and disgust, and it becomes necessary to relieve her of the annoyance by removing the book. Now it is found that a particular word has been the source of the excitement. Nothing in the word itself could explain the effect which it produces. A complete knowledge of her history, however, unravels the mystery. The explanation is this: a compound word happens to contain the syllable which is the name of an individual to whom many years ago she took a strong aversion, although she had little acquaintance with him. She would never enter the room where he was. It is long since he died, but she still dislikes to see or hear any name that contains his, even although in part. This aversion has ramified so completely into her life that it is sometimes very difficult to trace a particular act to it, but there is reason to think that it is always there. It may be traced in the idea of dangerous contamination with certain objects. She objects to people whom she meets touching her, and this on careful sifting turns out to be part of the same irresistible dislike to the individual in question. Hence the

demand she makes to have certain things washed. When she comes in from a walk she invariably washes her hands and face. She is most particular in washing away dust. Frantic washing of hands and arms is sometimes preceded by maledictory expressions. Occasionally words come into her mind in this wise: she will startle a friend by saying "verdigris," and then she will say in an appealing manner: "Thinking of it won't make verdigris come, will it?" She will also ask questions again and again which bear on her fear of contamination, saying, "Let me say it again; did so and so touch my dress?"

We see then in this case an illustration of the fear of contamination with objects, in themselves absolutely clean, but associated by some accidental circumstance with an individual or a long past event in the patient's life.

As to her family history, there is no proof of insanity or epilepsy on either her father's or mother's side; both families have been healthy and long lived. A cousin, however, is almost an idiot. An uncle was paralysed. The patient herself had a very severe illness in her infancy, but her childhood had passed when the unreasonable aversion to the gentleman had developed. In character she is impulsive, affectionate, generous in thought, but selfish in action, and with considerable religious feeling and sense of duty, not always carried out into practice. She is exceedingly truthful and unsuspicious. Intellectually she is regarded in society as perfectly sane, exceptional in wit, fond of science and music, and possessed of an excellent memory, with however, a great lack of patient application. There are no hallucinations of any of the senses.

I now pass on to describe the symptoms of a very striking example of imperative ideas assuming the form of Charcot and Magnan's onomatomania, and the malady of doubt in general.

(14) A boy of 19 (sent to me by Dr. Barlow), a law student who had gained distinction at school, and matriculated at the London University, met in the course of his reading with the expression, "*It was not incompatible.*" Shortly afterwards he came across the German words, "*Ich liebe es nicht.*" Now, it struck him that the negative in the former sentence preceded, and in the latter succeeded the most important word. He then began to puzzle himself about negatives in general. Whatever he read raised the question in his mind as to the construction of sentences in which a negative occurred. It became an all-

important, an absorbing problem. It interferred with his reading and working. The burden of his life was to place the negative in its right order, whatever that might be. One day he said to his father, "If it wasn't for that confounded negative I could master Blackstone and any other law book." At one time he was puzzling himself over the question, "Why haven't *we* cold blood, like some animals?"

The great danger is that he will become irresolute and vacillating in his actions, the continuance of the original mental difficulty weakening his will power and threatening to paralyse practical life altogether by the number of questions and doubts which arise in his mind as to the possible course to take in a particular instance. The proverbial three courses will not suffice long for those who become the sport of innumerable doubts and endless questionings. The law student's career becomes itself "a negative," in the presence of his morbid fascination for negatives.

With regard to heredity, I find that his father, a man of fine physique, is subject to attacks of mental depression, while the young man's maternal uncle has been for many years an epileptic.

Indeed, we cannot fail to be struck with the frequent occurrence of epilepsy in the families of persons labouring under imperative ideas. Napoleon, himself an epileptic, could not help counting the number of windows in the houses he passed. At certain points these cases seem to be closely allied to epilepsy. Now, it would be felt quite inappropriate to apply the term insanity to such a case as that of the law student. We must, indeed, admit that we can hardly regard the individual who cannot control his thoughts in the way that he wishes to control and direct them as a person of perfectly sound mind; but no "last will and testament" would be set aside in this case, or on the ground that a testator had an invincible desire to touch certain objects, or an intense repugnance to touch certain articles of furniture, or that he had an intolerable fascination for a particular word, or that certain ideas were associated with a special dress with which no one else had the same feeling. Nor would such morbid habits of thought and association of ideas go far to sustain the plea of insanity and irresponsibility in a criminal trial.

I now come to the last section of my paper:—

Cerebral Conditions Accompanying Imperative Ideas.

If now we consider what are the correlative changes in the cortex of the brain, we must say at once that no microscopical examination would disclose any pathological lesion associated with the morbid mental condition I have described. We are assisted, however, by starting with Laycock's doctrine of the reflex or automatic function of the cerebral cortex, as enunciated by him in a paper written fifty years ago. In this very original paper, read at York in 1844, before the British Association, he says of certain morbid mental conditions, that "their detection is sometimes difficult, because the patient being keenly conscious of his infirmities will conceal them. If, however, by what is termed the association of ideas, the morbid action of vesicular neurine be brought within the current of his thoughts, he becomes utterly powerless to resist it, as much so as the electro-biologised (hypnotised) to resist the suggestions presented to their minds. The formation of these *substrata* is due to the fixity of the mind on one idea or class of ideas, at a time when, from morbid changes induced in the vesicular neurine (as by undue mental labour, intense emotional excitement, want of repose, the development of a dormant predisposition, and the like), it is unusually susceptible to the operation of the unconsciously constructing mind, so that the fixed ideas become deeply writ, as it were, on the vesicular neurine, in the same way as acquired instincts, habits, &c.; and are, in fact, as difficult to remove.

Laycock writes: "The principle of reflex cerebral action will be found to be of the utmost importance to the elucidation of the relation of thought and will to the encephalon and to the comprehension of the marked changes they and it undergo in all forms of insanity."

In explaining some instances of imperative ideas by accidental association, it will very often be found that this association has arisen from their contiguity under some powerful emotional impression. I know a physician who cannot think of his father's funeral, which occurred some forty years ago, without the troublesome mental image of a white terrier being, to his annoyance, called up, simply because one appeared during the ceremony when the coffin was about to be lowered to the grave. Clearly this is due to the vividness with which the incongruous circumstance was at the moment registered upon his cortical tabula, never to be erased.

Laycock taught that "there is not only cerebral evolution in regard to healthy brain development, but an equally general inverse law of *dis*-volution (*sic*), equally important in studying mental disorders."

Laycock's theory fits in with the latent influence of association, not only of ideas, but of certain impressions received through the special senses, for in some forms of imperative ideas these become accidentally and sequentially connected in an extraordinary manner. The individual wishes to destroy this newly formed and accidental association, but he becomes absolutely dominated by it. Laycock, in his now little read article, insists that "the true explanation of the association of ideas is to be found in the doctrine of the reflex functions of the brain" (*B. & F. Med. Review*, January, 1845, p. 311), and yet, strange to say, no one except Dr. Jackson has done him justice, many have not once referred to him, and some have not heard of him!

Laycock recognised the control exercised by the cerebral cortex over sensory impressions, but does not appear to have anticipated current doctrines in regard to the control of one portion of the cortex over another, or positive and negative conditions of the mental functions. These doctrines it remained for Anstie, Monro, and above all Hughlings Jackson (who always expresses his indebtedness to them, and to Herbert Spencer), to teach.

I believe that Dr. Jackson has not in any of his writings referred specifically to imperative ideas other than as insane delusions, which according to him "signify evolution going on in the remains of a mutilated nervous system, as certainly as the patient's belief, when sane, signified evolution going on in the entire (*i.e.*) unmutilated nervous system." (Leeds Address by Dr. Jackson, 1889.) There is in short a "disvolution" of the highest cerebral centres, but as Dr. Jackson points out "there are all gradations traceable as a consequence of disease beginning in the highest centres, from such slight depths of dissolution as those which permit nearly normal actions, to dissolutions so deep as to afford no evidence of mentation." (*Med. Press and Circular*, December 9, 1871.)

Accepting this hypothesis, in addition to Laycock's great fundamental doctrine of the reflex or automatic function of the whole cortex, we seem to find an answer to the question I began with, what are the cerebral conditions accompanying imperative ideas? The most automatic is no longer under the control of the voluntary or least automatic cerebral levels or layers, or in Jacksonese, "reversals of evolution have occurred, being reductions from the least to the most organized of the highest cerebral centres."

Take the simplest and most frequent illustration of imperative ideas. Some lines of poetry come unbidden into the mind. There is nothing remarkable in that. They recur half-a-dozen times in the course of the morning. Nor is there anything very remarkable in that. If, however, they recur again and again during the rest of the day, the limit of healthy cerebral action may have been passed, and the individual may discover two things, *first*, that when a word in one of the lines presents itself, he has no peace or relief until they are all mentally (and perhaps verbally) completed, and *secondly*, that to his annoyance the relief is only short-lived, and the same obligation as before soon arises and tyrannises over him. What at first was a pleasure has become a nuisance. Here then is the earliest stage of an imperative act of memory. In the majority of cases a good dinner, a good night, and exercise in the open air will break the spell by reinforcing inhibitory power. But in others the automatic process returns and is as inevitable in its action as the going off of an alarum. Worst of all, words now present themselves, and recur again and again, which not only harass by their persistent frequency, but by their unsavoury character. They do not, or may not, find vocal expression at first, but sooner or later a motor discharge takes place which is irresistible.

With regard to those cases in which there is a morbid *dread of dirt,* I do not know that we can say more, in many instances, than that there is an exaggeration of the scrupulous cleanliness which in a marked degree characterises some persons who are in perfect health, but the origin of the imperative idea may occasionally be traced to some affection of the skin which has necessitated the attention being drawn to it, and has at last induced a morbid state of mind, not of introspection, but of "extero-spection." Should the general mental power be lowered, or a hereditary predisposition exist, this tendency to attend to the state of the skin becomes a passion, and results in the needless and wearisome washings of the body or the dress. Indeed, it too often happens that at last the patient no longer perceives the folly of these ablutions, but labours under an actual delusion.

With regard to the singular habit of *touching* certain objects without apparent reason, it is sometimes explained (by persons labouring under it) as due to a fear lest something will happen if they do not do it; but in other cases as an irrepressible habit without any motive whatever, often performed almost unconsciously as well as automatically. There may, however, be a conscious struggle between the voluntary or least automatic, and the most automatic cerebral action, the former being too feeble to control the latter. It often happens that ideas of touching are associated with ideas of doubt. In fact "doubting Thomas" touches in order to satisfy his doubts.

Among the illustrations I have given of imperative ideas, the most curious, perhaps, is that of automatic counting. And yet the act of counting is one so peculiarly automatic that it is natural it should be performed by persons with weakened higher centres. We have seen how in the case of a lady, she likened her state to that of the boy who is assisted in running a race by hearing or uttering numbers before making the start.

Of the malady of doubt I have said that a troublesome, although slight degree of it, is not uncommon in perfectly sane people. It was only the other day that an officer of an asylum engaged in laboratory work, told me that not unfrequently after shutting the door the last thing at night and having no doubt that it was shut, he would return once or perhaps twice to satisfy himself that this was the case. In the same way I have known persons open an envelope in which they had placed a cheque and had exercised great care that the date and signature were correct, in order to satisfy themselves that they were so. And again, a man may give to a friend, verbally, the number of a particular street quite correctly, and have no hesitation about it, but within five minutes may have misgivings, visualise another number, and begin to fear that he has said 20 when he ought to have said 40. Is this because the registration of the number has been feeble? This seems hardly likely, because one has noticed these doubts arise in the prime of life, when the brain would receive impressions clearly and retain them, as well as in persons more advanced in age.

I once knew a gentleman, tall and massive in form, who developed an imperative idea, the origin of which was clear enough. He began by thinking that if he died when from home, this might happen in a house in which it would be impossible for the undertaker to get his coffin down the stairs. The idea after a time haunted him and became very troublesome, and moreover, he said, that at last the worry was not confined to himself, and that he never met a man of tall stature without perplexing himself with the question whether his staircase would allow of his coffin being taken down stairs. I may add that a son and a grandson committed suicide. It cannot be said that Bacon's axiom, "He that questioneth much shall learn much," receives confirmation from these cases, for in a man's questioning thus, his last state is worse than the first.

What strikes one forcibly in regard to most, if not all, imperative ideas is that between them and ordinary ideas the difference is one of degree, and that it is a most difficult thing to determine when the boundary line has been passed. This on the psychological side. And on the physical side, what we want above all things to know is what cerebral change, degeneration, or dissolution has occurred by which the man who to-day is conscious of and smiles at an imperative idea, gravely believes it to-morrow, and labours under a systematised delusion.

Granting that there are sub-levels and superimposed levels in the highest centres themselves, we must, I suppose, assume that in the case of imperative conceptions without actual insanity, no pathological changes have induced dissolution of the very highest level or area, and that this only happens later on, when the patient with more extended dissolution no longer distinguishes between true and false ideas. The predominating idea has, in fact, become part of his own being, and not one extraneous to himself upon which he can look down as distinct from his personality—his ego.

In short and finally, is the explanation to be found in the degree or extent of dissolution in the same range or level of the highest centres, or is it to be found in different depths of dissolution in these centres? [1]

---

[1] Those who are familiar with German and French writers, will find that their recognition of the mental phenomena described in this article has been distinctly clearer than our own.

ON THE ASSOCIATION OF DISEASE OF THE SPINAL CORD WITH PERNICIOUS ANÆMIA.

BY H. M. BOWMAN, M.D.

ATTENTION has recently been directed by certain German observers to the fact that in patients suffering from severe progressive anæmia, of the type usually known as "pernicious," symptoms now and then develop pointing to an affection of the spinal cord; and it has been shown that these symptoms, though varying somewhat in some respects, are in others fairly constant, and are associated with changes in the cord of such striking similarity as to justify the cases being regarded as belonging to the same pathological group.

This association of severe anæmia with lesions of the spinal cord was first pointed out in 1887 by Lichtheim, who described three instances which he had observed, and he laid stress upon the special character of the changes found in the cord as distinguishing these cases from tabes, to which there was some general clinical resemblance. These cases were subsequently published in detail by Minnich,[1] together with three others which he had since observed. In these six cases, which were characterised by the usual symptoms of pernicious anæmia, the nervous phenomena bore a distant resemblance to tabes in some instances, in others rather suggested disseminated sclerosis, the most constantly observed being a progressive muscular weakness, which was found in every case, and subjective disturbances of sensation in the extremities which were twice wanting. Ataxy was noted in half the number, but was associated with loss of the knee-jerk in one case only, which was also unique in the fact that the pupil had lost its reaction to light. The gait, in most cases, could not be accurately noted owing to the marked motor weakness. Objective sensory disturbances, varying between partial anæsthesia to all forms of stimuli and slight disturbance of sensibility to one or more forms only, were observed in five cases, and were most marked in the legs. The condition of the knee-jerk varied considerably, it being reported to be absent twice, diminished once, normal once, and exaggerated twice. Involuntary muscular spasms occurred twice. Loss of control over the sphincters was present toward the end in half the cases. All these cases were males at about middle life, and in all the fatal termination occurred within a year of the first development of the nervous symptoms. The degenerations found in the spinal cord showed some difference in regard to extent, but there was close agreement in the distribution. In every case there was degeneration in the posterior columns, most marked in Goll's tract, but also involving more or less of the postero-external columns, but in the latter case leaving a strip of undegenerated white matter on the inner surface of the posterior gray cornua. The posterior roots and Lissauer's tract were invariably intact. In four instances the lateral columns were also affected, the degeneration being situated chiefly in the outer and posterior portion, and in three of these cases similar changes were also observed in the anterior columns, chiefly in the situation of the direct pyramidal tract. It was pointed out that in spite of the marked degeneration in the posterior columns there was never found to be any shrinking such as occurs in tabes, which may probably be explained by the nature of the lesion, which rather suggested a subacute process than a chronic sclerosis.

---

[1] *Zeitschrift für Klin. Medicin*, xxi., 1892.

Von Noorden[1] published another instance in a woman of 59, who was suffering from progressive anæmia, and in the course of this disease developed spinal symptoms, indicated by weakness and paræsthesiæ of all the extremities, impaired sensibility to touch in the legs, loss of the knee-jerk, and towards the end disturbances of the sphincters. The pupil reactions were normal, and ataxy was not obvious. The spinal cord showed "total degeneration" of the posterior

[1] *Charité Annalen*, 1891.

columns, not involving the posterior roots, and degeneration in the crossed pyramidal tracts.

In a case described by Eisenlohr[1] as an example of pernicous anæmia probably due to atrophic changes in the gastro-intestinal tract, were noticed motor weakness and paræsthesiæ in the extremities, a spastic condition of the legs, loss of knee-jerk without obvious ataxy or affection of the pupil, and, later, loss of control over the sphincters. He found degeneration in the posterior columns of the cord, closely resembling that usually occurring in tabes, as well as scattered foci in the lateral columns. His patient was a man aged 59.

Two interesting cases have recently been recorded by Nonne[2]; the first being a man aged 48, who, while suffering from pernicious anæmia, developed nervous symptoms characterised by numbness and weakness in the lower extremities, definite ataxy in all limbs, occasional girdle sensation and spasms in the legs, normal pupil reactions, slight transient analgesia in the legs and, later, loss of control over the sphincters. The knee-jerk varied; at first it was diminished, then was entirely lost, and later was again distinctly obtained.

His second case, a man aged 57, had suffered from uncertainty in walking, occasional girdle feeling, and numbness in the hands and feet for some months *before* anæmia was noted. At first he had ataxic gait, slight anæsthesia and analgesia of the legs and fingers, which ultimately cleared up, and greatly diminished knee-jerks. Later, anæmia had become very prominent, the knee-jerks could be readily obtained, and motor weakness had increased, ataxy being no longer demonstrable, and there was incontinence of urine. The light reflex of the pupil was never lost.

In both these instances the changes found in the cord were most marked in the posterior columns, especially in Goll's tract, which presented the appearances usually found in ascending degenerations in the upper dorsal and cervical regions. The postero-external columns showed a variable degree of change, but everywhere the zone bordering the

[1] *Deut. Medicin. Wochenschrift*, 1892.
[2] *Archiv für Psychiatrie*, xxv., 1893.

inner aspect of the posterior cornua, the posterior nerve roots, and Lissauer's tract were unaffected. The lateral columns were extensively involved in the region of the direct cerebellar and crossed pyramidal tracts, and there was slight degeneration in the direct pyramidal tracts. In the anterior, and partly in the lateral, columns the process was found to consist in the more or less complete coalescence of small dotted foci of degeneration, apparently of comparatively recent formation.

The case which I will now describe was recently in the National Hospital, under the care of Dr. Hughlings Jackson, to whose kindness I am indebted for this opportunity of publishing it. In the main points of this case, both clinical and pathological, will be found a close resemblance to the ten cases alluded to above, especially, perhaps, to the two described by Nonne, and it must clearly be included in the same group.

Elizabeth R., aged 53, married, three children, was admitted on July 12, 1892. Sallowness was first noticed two years previously, during which time she had also suffered from headache, and recently she had become much paler. Nine months ago the legs began to feel very weak, and for one month a feeling of "pins and needles" was constantly present in the legs.

Her previous health had been good. There was nothing pointing to syphilis beyond a single miscarriage. Menopause a year ago, but severe "flooding" two months before admission.

On admission the mucous membranes and skin were very anæmic, the latter having a lemon yellow tint. General nutrition fair, there being abundant, though not excessive, subcutaneous fat. She was unable to raise her head from the pillow without giddiness. Appetite fair, bowels acting regularly, tongue clean. Urine non-albuminous. The lungs presented no evidence of disease. The heart was not dilated, and no hæmic bruit was audible. Liver dulness commenced at the sixth rib, and the edge was felt more than an inch below the costal arch. The spleen just descended below the ribs on deep inspiration.

Nervous System.—All voluntary movements of the trunk and extremities could be performed, though somewhat feebly, no actual paralysis being demonstrable. From patients being unable to stand the gait could not be observed, but there was no ataxy or tremor in the movements of the limbs.

Sensibility was affected as follows :—There was blunting to very light tactile and painful stimuli in the finger tips, and slight tactile anæsthesia in the legs below the knees, analgesia being less evident and confined to the toes.

The knee-jerks were decidedly brisk, wrist-jerks were readily obtained, but no ankle clonus could be elicited. The plantar and abdominal reflexes were brisk.

No affection of the cranial nerves was found.

The eyes were free from nystagmus ; the pupil reactions normal; distant vision good, though accommodation was weak ; retinal hæmorrhages of various sizes were found in the fundus, as well as some white patches of effusion.

Examination of the blood showed the red cells to be 19·5 per cent., and hæmoglobin 20 per cent. of normal ; micro- and poikilocytes were found, but no large nucleated red cells, or "eosinophil" cells were discovered (these were never found at any subsequent time) ; white cells were not increased.

The mental condition was unaffected. Control over the sphincters was complete. No girdle feeling or pains in the limbs were complained of.

Treatment by iron having been tried for a time during which the anæmia gradually became worse, arsenic was substituted with apparently striking results, as will be seen below.

August 22.—Slight improvement in general condition, anæmia being less intense. Blood estimation showed an increase of red cells to 32 per cent. of normal. Since admission the temperature had remained about normal, only occasionally reaching 100·4, the maximum observed. Less motor weakness. Sensation practically unaltered, but it was noted that there was distinct impairment of position sense in the toes, most noticeably at the big toe joints. Localisation of stimuli was correct. Subjective sensations of "pins and needles" still complained of in the hands and legs. Reflexes unaltered.

September 21.—Marked improvement in general condition, anæmia being much less intense, and patient was able to sit up out of bed, being able to walk a short distance with the help of a nurse, or in a "go-cart." No ataxy was observed when she stood up or walked. Sensibility was greatly improved, there being no impairment to tactile or painful stimuli, though a touch had a "numb" feeling on the feet and legs. The blood value had increased to 58 per cent. of normal.

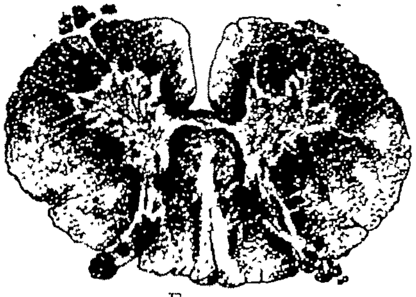


Fig. 1.

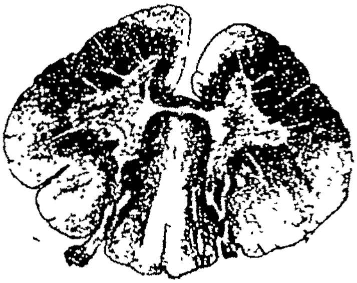


Fig. 2.

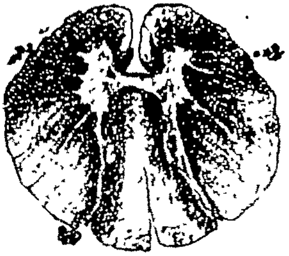


Fig. 3.

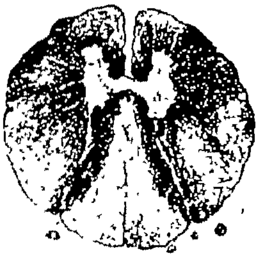


Fig. 4.

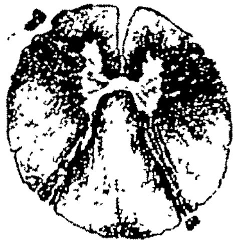


Fig. 5.

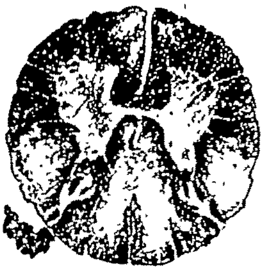


Fig. 6.

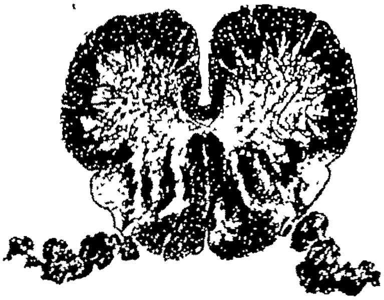


Fig. 7.

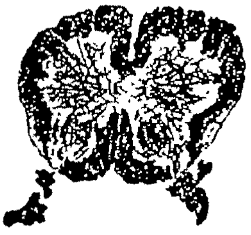


Fig. 8.

October 7.—Still further improvement. The blood reached about 82 per cent. of its normal value in red cells. No hæmorrhages were found in the fundus oculi.

October 29.—Patient was now able to walk without support, her gait, beyond a certain amount of feebleness, showing no special alteration from health. Sensation normal. Spleen not palpable now. Complexion was rather sallow, though anæmia was not noticeable. Blood estimation gave proportion of red cells at 87 per cent.

Patient was soon after this sent out to a convalescent home, and then was kept under treatment as an out-patient. Early in 1893, however, she was found to be worse again, and on March 3 she was re-admitted, having been unable to walk for a month, and giving a history of occasional retention of urine. At the time of re-admission her general condition was very much the same as was noted when first she came under treatment, there being marked anæmia, with a sallow tint of skin, but no evidence of disease of the chief thoracic and abdominal viscera. The spleen could not be felt.

Examination of the nervous system gave the following results :—She was quite unable to stand, being only just able to raise her body into a sitting posture by great effort. Movements of the arms were rather feeble; those of the legs markedly so, there being just power of raising the heel from the bed. On executing fine movements with closed eyes slight ataxy was found; no tremor present. The legs were subject to sudden and painful involuntary spasms.

Sensation was considerably disturbed, there being impaired sensibility to touch, pain, and temperature in the lower limbs, most definite towards the feet, and gradually fading towards the trunk, which was only slightly affected. Localisation was very incorrect, and position sense was completely lost at the toe, ankle and knee joints.

Reflexes were in the same condition as was observed in the first note.

The sphincters were unaffected. The *blood* showed red cells in the proportion of 68 per cent.

July 23.—Much weaker and more anæmic. Blood estimation gave 37 per cent. of red cells. She could not turn over in bed unassisted, arm movements were feeble, leg movements markedly so. Ataxy was now very definite in the arms. The legs were semiflexed and rigid, passive extension causing great pain, and spasmodic jerkings were more painful and frequent. The impairment of sensibility was more definite, and was found on the fingers for the first time. Knee-jerks very brisk; a non-persistent slow clonus obtained at the ankle; superficial reflexes exaggerated. Pupil reactions normal. No retinal hæmorrhages.

August 11.—Was much worse. Vomiting was troublesome, and interfered with medicinal treatment. The temperature was constantly above normal, frequently reaching 101°. Retention, followed by incontinence of urine, and later of fæces, was observed. There was œdema of the feet. Blood value about 25 per cent. of normal.

September 5.—Patient was now quite helpless, being unable even to put her arms out of bed without help. There was wandering of the mind at night. Incontinence was complete. Œdema of the legs extended to the knees. Anæmia was very marked, red cells being in proportion of 18 per cent.

September 13.—Extreme anæmia—red cells being reduced to 12 per cent. Patient constantly in condition of muttering delirium. Temperature constantly subnormal. Both eyes showed numerous retinal hæmorrhages. Death occurred on the 16th.

At the autopsy there was great anæmia of all viscera. The pleuræ contained free fluid, and the bases of the lungs were congested. The heart was pale, but not obviously fatty. The liver large, pale, and fairly firm—no free iron reaction was obtained either on fresh section or in stained specimens. The spleen was not enlarged. There was a cyst communicating with the duodenum, apparently of inflamatory origin, situated in the head of the pancreas. In the fresh cord a gray discolouration was seen on section, occupying the posterior columns, and most marked in the upper dorsal region. Otherwise the cord and brain appeared normal, except for extreme pallor.

After hardening in Müller's fluid a very extensive degenerative process was apparent in the cord, involving not only the posterior columns, but also more or less of the lateral and anterior columns. Sections were stained by the Pal-Weigert method, and in carmine, and under a low power there was found to be the following distribution of the lesions :—

(1) *Upper Sacral.*—The degeneration is here limited to a small patch at the outer and posterior margin of the lateral columns, which is more extensive and more complete on the right side, though on neither side is the process far advanced (see fig 8).[1]

---

[1] In this, and in all the other figures, the degeneration has been made to appear slightly more intense than is actually the case.

(2) *Mid-lumbar.*—In the lateral column of either side is a patch of degeneration occupying the posterior portion of the column, and presenting a roughly triangular outline, with the base, where the change is most complete, extending to the periphery of the cord. On all other aspects the degenerated area is surrounded by healthy white matter, except at the extreme posterior extremity.

In the posterior column the only apparent change is in the form of an irregular collection of foci at the base of the right tract of Goll (see fig. 7).

(3) *Upper lumbar.*—In each lateral column is a patch of degeneration having a similar distribution to the above section, except in being of larger extent, and in the densest degeneration being in the centre of the area. Except at the outer side there is healthy white matter on every aspect into which the degenerated portion of the column gradually merges.

In the posterior columns changes are found occupying the greater portion of the tracts of Goll, the most complete change being in the centre of each area at the margins of which it becomes patchy in distribution and fades gradually into healthy tissue. The base of the tract is unaffected. In the postero-external columns the changes are slight, and chiefly confined to the basal part, being most marked on the left side.

Small foci of degeneration occur in the direct pyramidal tracts especially in the left (see fig. 6).

(4) *Lower dorsal.*—The lateral columns are here very extensively involved, the healthy tissue, into which the degeneration very gradually fades, being considerably the smaller part of the columns.

The greater part of the posterior columns is also involved, the healthy white matter being limited to a narrow strip bordering the inner aspect of the posterior cornua and the gray commissure, and a wedge-shaped area at the base of the median fissure. Scattered islets of partly degenerated white matter are found in the affected tissue, though for the most part the change is very complete.

In the anterior columns degeneration is found in the direct pyramidal tracts, where the change is most advanced, but it also extends outwards along the margin of the column for some distance, gradually fading and assuming a dotted distribution (see fig. 5).

(5) *Mid-dorsal.*—In most respects the changes here are similar to those in the last section, though they are more complete and more uniform, and there are fewer healthy fibres at the base of Goll's tract.

There is a small island of degeneration in the left antero-lateral tract (see fig. 4).

(6) *Upper dorsal.*—The changes in the lateral columns, though still involving a large area, are now rather less extensive, and less complete, except toward the periphery of the cord. In general the distribution is similar to that described above.

In the posterior columns the degeneration closely resembles a secondary ascending process in distribution, being almost confined to Goll's tract. The changes extend into the inner aspect of the postero-external columns, which are also affected slightly at their bases.

The anterior columns are chiefly affected at their median and anterior margins. The antero-lateral tracts are free (see fig. 3).

(7) *Lower cervical.*—In the lateral columns the greater part of the white matter is still involved, though the affected area is dotted here and there with islets of comparatively healthy fibres, and is for the greater part less completely changed. The distribution is principally marginal.

The posterior columns show very dense changes, pretty sharply mapping out the limits of Goll's tract, which, except at the base and near the commissure is almost completely degenerated. The postero-external columns present slight changes along their inner aspect, and more advanced changes near their bases, especially on the left side.

The anterior columns are affected to the same extent as in the last section (see fig. 2).

(8) *Mid-cervical.*—There is less degeneration in the lateral columns, the peripheral region being the part most affected, and the changes are generally much less advanced.

In the posterior columns Goll's tract is almost entirely affected, and the changes are very advanced. The outer columns are more extensively involved at their bases than in the last section, though here, as in every other instance, the degenerated areas are separated from the posterior gray cornua by a strip of healthy white matter. On the right side there is also a collection of foci of degeneration at the anterior part of the column.

The anterior columns are affected much as in the last, though the change is almost limited to their median aspect (see fig. 1).

(9) *At the lower part of the decussation* there is a wedge-shaped area of degeneration occupying the posterior column, chiefly in the postero-median portion. In each lateral column is a small peripheral area which has undergone a similar change. The pyramidal fibres are affected in a somewhat irregular manner, and degenerated fibres can be detected in the decussation.

(10) *Just above the decussation* the changes are found to have a similar distribution, but are less extensive, and less complete. In the pyramidal tracts the degeneration has a dotted distribution.

In no part of the cord can any shrinking of the affected columns be detected.

Under a higher power of the microscope the degenerative process is found to vary considerably in the different regions. Taking the periphery of an affected area in the anterior or lateral columns, where the change is least dense, and may fairly be assumed to be in an early stage, the most obvious alteration found in the nerve fibres themselves. At first a few isolated fibres may be seen among apparently healthy structures, the medullary sheaths of which are swollen, often to three or four times the diameter of a healthy fibre, and which take Weigert's stain less readily, so as to appear paler than usual; the axis cylinder still appearing to be normal. Among such fibres are found several empty spaces where the affected fibres have dropped out, probably in the cutting and staining processes. The interstitial tissue in such situations does not show any very obvious change.

On examining a part where the degeneration is apparently more advanced it is found that the proportion of healthy fibres is much smaller, the majority presenting some degree of the changes mentioned above, extreme degrees of swelling of the medullary sheaths being shown, and from the relative diminution in the number of fibres a larger proportion have probably been destroyed or have dropped out of the section. The interstitial tissue is somewhat thickened, and a number of nuclei are seen scattered about between the nervous elements.

In the parts of the section where the most intense changes have taken place, it is found that the interstitial tissue is largely in excess, such nerve fibres as are present being isolated and presenting more or less swelling of their myelin, while a considerable number of nuclei can be seen here and there. In the upper part of the cord a still greater increase of connective tissue elements is found in Goll's tract, where nerve fibres are in places entirely wanting, and here the interstitial tissue has a denser appearance than elsewhere.

The blood vessels are found to be affected in proportion to the degree of interstitial change, being apparently healthy in areas where the nerve elements are solely or chiefly involved, and presenting very obvious changes in the more densely degenerated parts: The earliest alteration consists in a proliferation of nuclei in the sheath of the smaller arterioles, while in a later stage the sheath is crammed with cells, the lumen of the vessel remaining normal. In the most advanced areas of degeneration (in Goll's tract) the walls of the arterioles are greatly thickened and the lumen much reduced, in some instances being occluded.

The posterior roots are everywhere intact. The posterior cornua are also free from change, though the degeneration in the lateral columns extends up to them at some levels. They are always bordered by a strip of healthy white matter on their inner surface.

The ganglion cells, the gray matter generally, and the pia mater are entirely free from change. The peripheral nerves, unfortunately, could not be examined.

From the above description it will be seen that the degenerative changes are for the most part not strictly limited to definite tracts of long fibres, also that appearances which may reasonably be regarded as indicating an early stage of the process are met with at every level of the cord, from the mid-lumbar to the highest cervical region. From these facts it may be inferred that the process is a primary one, affecting all three columns, though at least the denser degeneration found in the situation of the crossed pyramidal tracts at the lower levels, and in Goll's tract above the mid-dorsal level are probably of the nature of secondary descending and ascending lesions respectively.

The minute changes suggest that the process is rather of a parenchymatous than an interstitial character, at least primarily.

I have to acknowledge my indebtedness to Dr. W. S. Colman for much kind help and advice in the pathological investigation of the cord.

Remarks.—Clinically it will be seen this case presents many points of resemblance to the group of cases previously referred to, especially in the gradual onset of nervous symptoms commencing with subjective sensory disturbances and progressive motor weakness, and in the comparatively short course from the first onset of these symptoms to the fatal termination; though in this one respect my case was somewhat unusual in its longer duration, which extended over a year and nine months, including, however, a period of three months, during which there was an almost complete freedom from symptoms.

In the later stages of these eleven cases the nervous phenomena presented considerable variations, a few cases (those of Lichtheim) bearing some resemblance to tabes, others rather approaching the type of disseminated sclerosis, and to this latter group my case belongs. The resemblance to disseminated is not at all close, as such important symptoms as volitional tremor, nystagmus, and articulatory changes were absent. A careful study of this group of cases does not reveal any one symptom or complex of symptoms which would justify their being regarded as forming a distinct clinical group, beyond the one fact that there was present, in addition to the nervous phenomena, an anæmia of severe and progressive type, and they must, I think, be included in the large and rather ill-defined class of "combined postero-lateral scleroses," the differences in the symptoms depending upon the relative extent of the lesion in the posterior and lateral columns respectively.

In his article on "Ataxic Paraplegia," Gowers[1] states that the disease is usually characterised, among other things, by a gradual onset, a slow course without special tendency to shorten life, and by an intact condition of sensibility even in the late stages, as well by an excessive activity of the deep reflexes. He adds, however, that in rare cases there may be sensory impairment, and in some the knee-jerk is lost. In these anæmic cases, on the other hand, the course was always rapid, being usually less than a year, sensory disturbance was observed in every instance, and the knee-jerk presented many variations, being absent throughout in four cases, and temporarily in two others, and in no instance was it present in great excess. Too much importance must not be attached to these differences, since many cases of postero-lateral sclerosis have now been recorded in which anæsthesiæ or loss of knee-jerk, or both, have been present. Probably the presence of the grave blood disease is quite sufficient to account for the rapid course of the anæmic cases, though a similar short duration of the disease has been more than once observed where no special allusion is made to the presence of anæmia (Strümpell,[1] Ormerod,[2] and others.) On these grounds, this group of anæmic cases may be taken to belong to the class of postero-lateral scleroses, so far as their clinical features are concerned.

---

[1] "Diseases of the Nervous System," 2nd edit., vol. i, p 453.

In considering the pathology of these cases two questions arise: (1) Are the lesions found to be constant? As regards the *extent* of the degeneration this is certainly not the case, as in two instances the change was found in the posterior columns only, in two others in the posterior and lateral columns, while in the remaining seven cases all three white columns of the cord were involved, and even in these the extent of degeneration varied. In every instance, however, the posterior columns were most extensively affected, so that this apparent difference may depend upon the duration and severity of the process. With regard to the distribution of the changes; if we neglect Eisenlohr's case in which the distribution was reported to be very similar to that usually found in tabes, and Von Noorden's, where there was "total degeneration" of the posterior columns, though leaving the posterior nerve roots intact, in all the remaining cases certain features are found to be constantly present. Thus the degeneration in the posterior columns is most advanced and most extensive in the tract of Goll, which, in the lower levels, is frequently affected alone, while the changes in the postero-lateral-columns are less complete, usually less extensive, and are most marked in the middle or upper levels of the cord. In every case, and throughout the whole length of the cords, a zone of healthy white matter is found bordering the inner aspect of the posterior cornua. The lesion in the lateral columns, while chiefly affecting the crossed pyramidal and direct cerebellar tracts is not limited to any definite systems of long fibres. The microscopical appearances appear to be similar in most respects.

(2) Are these changes to be regarded as specific? This must be answered in the negative, since the distribution of the lesions is precisely similar to that found in numbers of cases of combined sclerosis, unassociated with anæmia, and

---

[1] *Archiv für Psychiatrie*, 1881.
[2] *St. Bart. Hosp. Reports*, xxix., 1894.

closely resembles that described by Gowers as typical of ataxic paraplegia. The microscopic changes bear many points of resemblance to the lesion of subacute myelitis, only in the most densely affected parts presenting any notable degree of sclerosis, and here the change is probably in great part of a secondary nature. Precisely similar changes have been observed in cases of postero-lateral sclerosis without anæmia, as in Ormerod's case alluded to above.

From the above considerations it will be seen that the pathological changes found in these cases cannot be regarded as in any way characteristic, which was also observed of the clinical course and symptoms.

It remains to be considered what is the relation between the anæmia and the spinal lesions. At first sight it seems tempting to assume that the degenerations in the cord are secondary to the grave blood state, and if this could be shown to be the case it would be a matter of some importance, as possibly throwing some light on the difficult question of the ætiology of some forms of spinal degeneration at least. Unfortunately this is not easy to prove. Minnich, in summing up his six cases, was disposed to doubt the dependence of the spinal disease upon the anæmia, since the latter was often of variable degree and sometimes of short duration, at the time of onset of spinal symptoms. He suggested that both trains of symptoms were dependent upon some unknown toxic influence. The same observer made an examination of twelve cords from patients who had died from pernicious anæmia without any nervous symptoms having been observed, and in five of them some changes were discovered, in some cases bearing a general resemblance to the lesions found in his six published cases. This is suggestive of there being some very close relation between the blood state and the spinal disease.

One argument against the anæmia being primary is to be found in the fact that in Nonne's second case the symptoms of spinal disease distinctly preceded the onset of anæmia.

On the other hand many points may be raised in favour of this view (that the spinal disease is the result, at least in some instances, of the blood state):—

First, it is seen that with one exception the anæmia dated back beyond the first observed spinal symptoms. In my case some degree of anæmia had been present for fifteen months before nervous symptoms occurred.

Secondly, it is worthy of remark that in my case during the gradual recovery of the patient under treatment the nervous symptoms also abated, until at the time of her discharge, when the condition of the blood was little below the normal standard, there was nothing that could be demonstrated beyond a slightly feeble gait and an exaggerated condition of the knee-jerk; but that, with the relapse of the anæmia, all the previous nervous phenomena returned and gradually increased with it up to the time of death.

Lastly, there is considerable evidence that in the course of anæmia of severe degree, no matter of what type, certain disturbances of the nervous system do now and then occur. On this question Gowers[1] alludes to the well-known fact that optic neuritis can occasionally be found in chlorosis, as an instance of the influence of general anæmia upon the nervous centres, and he mentions weakness of the legs, sometimes amounting to complete paralysis as another rare symptom. In anæmia from loss of blood, also, he describes paraplegia, usually affecting the motor functions only, as being sometimes observed. Through the courtesy of Dr. Buzzard I am enabled to refer to a case (as yet unpublished) which recently came under his care at the National Hospital.

The patient, a young girl, was the subject of extreme chlorosis, and when first observed there was found to be complete absence of the knee-jerks, even on employment of the "reinforcement method." During the course of treatment by iron, however, the jerks gradually returned, and they could be obtained without difficulty when convalescence was completed.

When it is considered that the blood changes and the general signs and symptoms in pernicious anæmia are usually much more grave than is commonly the case in chlorosis, or simple anæmia, it is only reasonable to expect that nervous phenomena, which are occasionally found in the latter conditions, should now and then be met with in the course of the former, and also that when they do occur they would be of a more severe type.

---

[1] *Loc cit*

The eleven cases were all of the type of pernicious anæmia, though probably the cause of this condition was different in a large proportion; thus two cases were ascribed to bothriocephali, three or four to gastro-intestinal disturbances, including one case where atrophy of the glands of the gastro-intestinal mucous membrane was found, and one case followed a metrorrhagia. In the remainder no probable cause was discovered.

In conclusion, I would suggest that these cases, in which spinal symptoms of a somewhat anomalous type are associated with grave and progressive anæmia, be regarded as belonging clinically and pathologically to the group of postero-lateral scleroses (ataxic paraplegia) and that until further light be thrown upon the subject it would be unwise to be too positive as to the relation of the nervous symptoms to the altered blood state, though I am inclined to regard the anæmia as primary, and probably the cause of the changes in the cord.

PATHOLOGY AND TREATMENT OF GRAVES' DISEASE.

BY JAMES J. PUTNAM, M.D.
Professor of Diseases of the Nervous System, Harvard Medical School.

GENTLEMEN,—The investigations and discussions with regard to the pathology and treatment of Graves' disease have reached a point that makes it possible to present several important issues of scientific and practical interest in a moderately definite form.

The essential questions are these: Are the symptoms of Graves' disease mainly toxic in origin, and due to an excessive or perverted secretion from a diseased thyroid gland; or does the affection belong to the class of the so-called neuroses; or, finally, are the symptoms due to an irritation of the thyroidal and cervical nerves?

If it can be determined that the thyroid is directly or indirectly the principal cause of the disease, then a strong case is made out for the removal of that gland by surgical means, provided no strong contra-indications are present. As a matter of fact, this operation has already been done in a large number of cases, and on the whole with encouraging results. The most successful results are, however, as we too well know, liable to be published first, and the proper position of thyroidectomy in the treatment of Graves' disease has yet to be established. My own experience with two cases has made me feel the need of caution in recommending this treatment.

The writer who first called attention to the importance of the thyroidal theory, and who has given it perhaps the strongest support, is Mœbius.[1] Other writers of prominence have skilfully maintained the same view, two of the latest of them being Dr. Greenfield, of London, who makes this topic the subject of the Bradshaw lecture of 1893,[1] and Rehu, of Frankfort,[2] who has had much practical experience with the surgical treatment of these cases.

---

[1] Schmidt's *Jahrb*, vol. ccx., July 15, 1886; *Zeitschr. für Nervenheilk.*, 1891; and other papers.

The argument for the thyroidal-secretion theory, drawn from the successful results of operations, is not as convincing as it at first sight seems. It happens often enough that morbid processes are kept up by influences which were not their real cause, and cease when these influences are withdrawn. Furthermore, the symptoms of Graves' disease have sometimes been favourably influenced by such operations as the draining of a cyst of the thyroid (Rehu), or removing a sarcoma (Tillaux) without doing much if anything to the secreting portions of the gland ; and operations for the removal of irritations from the nasal cavity have also been remarkably effective in a few cases in giving the first impulse toward cure.

The thyroidal theory has taken, in general, one of two forms : (*a*) That the amount of the thyroid secretion is increased, and, being poured into the blood, acts as a poison to the nervous system, and perhaps to other tissues ; (*b*) That the thyroid secretion, whether increased or not in quantity, is altered in character (v. Mœbius). The latter view was proposed to meet the objection that some of the symptoms of Graves' disease may appear before any recognisable glandular enlargement has occurred. The idea of an altered secretion is ingenious and suggestive, but is not yet substantiated. A great deal of careful chemical and physiological work must be done before we can decide such a question. Greenfield says that the secretion of the diseased gland is liable to become mucinoid, instead of colloid, in character, but this does not greatly help us. Now that Graves' disease is more commonly treated by thyroidectomy than formerly, it should be possible to obtain fresh material for direct experimentation, but until this has been done we ought to admit that we know simply nothing of the action of perverted thyroid secretion. It is difficult to believe that just the same perversion would occur in each case, always giving rise to much the same symptoms, *i.e.*, the symptoms of the typical Graves' disease, and it would not be proper to accept the arguments adduced in favour of the increased secretion theory as necessarily supporting the perverted secretion theory. It has also been suggested as possible (Maude, *Lancet*, 1893), that even when no obvious enlargement of the thyroid has occurred the secretion may be increased in quantity, but before much weight is attached to this view, as yet unverified, the validity of the thyroid theory in general deserves further scrutiny.

---

[1] *Lancet*, December 9, 1893.

[2] *Deutsche med. Wochenschrift*, 1894, p. 265.

(1) It is pointed out that Graves' disease presents various points of similarity or antithesis to the myxœdematous cachexia which follows the removal of the thyroid. Thus, in the first stages of this cachexia, symptoms of nervous excitability make their appearance, which are not unlike some of those characteristic of exophthalmic goitre. The temperature may be elevated, tremors are often present, and muscular jerkings sometimes occurring in broken series.

(2) Besides these points of resemblance, there are also points of contrast which are very marked at first sight—though, perhaps, they are in reality rather superficial—between the symptoms of Graves' disease and those of the later stages of cachexia thyreopriva, or the ordinary condition of a myxœdematous patient. This contrast is tersely expressed by Mœbius,[1] who says: "In the one case we have enlargement, in the other diminution in the size of the thyroid gland; in the one a rapid, in the other a slow heart beat; in the one we have a fine skin, warmer than usual and inclined to sweat, while in the other the skin is thick, cold, and dry; on the one hand we see increased mental irritability, a condition of irritable weakness, on the other slowness and dulness of the mind."

It is certainly obvious that in these two conditions we have symptoms which are in a measure the antithesis of each other, but this fact should not be admitted as indicating more than it really does indicate. Many of the strictly nervous symptoms of Graves' disease, such as the tremor, irritability, increased heart beat, and flushing of the skin, are met with in conditions of nervous excitement where no question of increase in the thyroid secretion comes in at all. As regards the differences in the condition of the skin, it is doubtful if there is any real contrast between the state induced by a relaxed vascular contraction and increased activity of the sweat glands, and that due to such manifest structural changes as are seen in myxœdema, where the diminution in perspiration is, for all we know, a secondary element. Fortunately, we have in the extracts of animal thyroids, now so largely in use for myxœdema and other states, a means of studying the action of the thyroid carefully and at leisure.

---

[1] *Zeitschr. für Nervenheilkunde*, 1891.

To some extent this has already been done, and the results seem, in some, though not in all respects, confirmatory of the thyroidal theory of the origin of Graves' disease. These facts have been passed in review by Murray (*Lancet*, London, 1893), Greenfield, and others. An excess of thyroid extract seems to cause, quite regularly, even in healthy persons, to judge from the few experiments reported, an increase in the pulse-rate and temperature, and often diuresis with increased secretion of urea. Myxœdematous patients are sometimes very susceptible to this substance, and sometimes suffer from serious prostration, with anginoid and neuralgic symptoms, as the result of a dose which other patients bear with ease.[1] In such cases, however, important changes in the metabolism partly related to the previous myxœdematous state, are rapidly going on, attended with great loss of weight, and it may be that these changes, or possibly the absorption of toxic products, are in part the cause of the symptoms noted. Loss of weight is, indeed, apt to occur even with healthy persons who are taking thyroid preparations, and yet it is noteworthy that patients with Graves' disease are not especially prone to emaciation as one might expect them to be if poisoned with thyroid secretion. It is obvious that the absorption of too much secretion or of an altered secretion might cause some of the symptoms of Graves' disease, but it cannot be said that anything like a clear case is made out in favour of the whole complex being due to that cause.

---

[1] See, for example, Shattuck: *Boston Medical and Surgical Journal*, 1894.

It has been said in this connection that thyroid extracts given to patients with Graves' disease seem as a rule rather to increase than to diminish the symptoms already present, but I do not consider this argument to be of much value. Inasmuch as the thyroid preparations increase the heart-beat in normal persons, it is obvious they would be likely to increase the tachycardia of Graves' disease, but even this is not a regular occurrence. I have been giving of late five to ten grains daily of desiccated thyroid to a patient with Graves' disease without any increase in her palpitation or nervous symptoms, and, in fact, with relief to some of them, and others have had the same experience.

A striking case of this kind has been published by Owen.[1] This physician attempted to treat a case of typical Graves' disease of twenty years' standing by the use of thyroid extracts. The patient's wife, by mistake, administered a quarter of a pound of chopped thyroid on each of two successive days. This was followed by nausea and eructations, vertigo, insomnia, swelling of the face and legs, but nothing of greater import. A week later the treatment was resumed, and a quarter of a lobe of sheep's gland was given daily, as originally ordered. Steady improvement soon began, which progressed so far that the pulse fell from 126 to 76 beats per minute, the exophthalmos almost disappeared, and the patient became able to do heavy work. A single fact like this is of great value, and seems to show that the improvement in the general nutrition which has now been shown to occasionally follow the use of the thyroid in non-myxœdematous cases may sometimes be made to serve a good turn in the treatment of Graves'[2] disease.

---

[1] *British Medical Journal*, Dec. 2, 1893.

[2] See a paper by the writer in the *Boston Medical and Surgical Journal*, February 15, 1894.

It occurred to me last winter while watching a case of thyroidectomy for Graves' disease,[1] that some of the ill effects of this operation may be due to the pouring out of a large quantity of thyroid secretion from the cut surface of the gland into the fresh wound of the neck. In this case a glairy substance actually exuded into the dressing. If the reports may be trusted these ill effects do not, to be sure, always occur, but in four cases (one of them a case of ordinary goitre) which have come under my observation, the patients were very ill, and the pulse very rapid. One of the patients died at the end of a week, though doing well in all respects except for restlessness, nausea, and cardiac weakness.

(3) Another argument adduced by Greenfield in favour of the thyroidal theory is that the histological examination of the thyroid shows that in Graves' disease we have evidence of an increase of the gland with signs of considerable activity. The thyroid of the adult is made up mainly of small and irregular spaces, lined with epithelium and containing colloid. In Graves' disease the structure becomes arborescent, suggesting that of a secreting gland, the epithelium becomes higher, and the colloid disappears or is changed into a substance of different character.

There is, however, another and quite different significance which may, with quite equal reason, be attached to these changes. It was pointed out several years ago by Halsted and Welch, of John Hopkins' Hospital,[2] that when the thyroid is mutilated, changes similar to those to which Greenfield has referred, regularly occur. In this case we are dealing with a regenerating gland-stump, but Canizzero[3] has found closely similar changes to occur as the result of slight injuries to the gland, such as were induced by separating it carefully from its attachments for the purpose of uniting it to the muscles of the neck. The histological changes occurring under these circumstances are characterised as a reversion towards the embryonic state. Perhaps they indicate an attempt at more active functionating, but more evidence of this is needed than we now possess. It is also noteworthy that even in phthisis changes perhaps analagous may occur.[1]

---

[1] Operation by Dr. J. C. Warren. See a paper by the writer in the *New York Journal of Nervous and Mental Diseases*, December, 1893.

[2] Unpublished communication to the Association of American Physicians; see *Transactions* for 1893.

[3] *Deut. med. Wochenschr.*, 1892, p. 184.

Again, it sometimes happens that Graves' disease gives place rapidly to myxœdema,[2] and this certainly looks as if the changes in the gland in Graves' disease were of a destructive tendency. Moreover, it is well-known that Graves' disease sometimes occurs gradually as a complication of ordinary goitre, and we have no evidence to show that under these circumstances any further increase in the secretion of the gland goes on.

Furthermore, if we may trust an observation by Sollier,[3] myxœdema, beside its liability to follow Graves' disease, or to occur in some other member of the family of a goitrous patient, may even co-exist in some measure with it.

Yet myxœdema can hardly be supposed to come on while the thyroid is secreting an excess of its active principle.

It is true that some of the symptoms of Graves' disease suggest the presence of some poison, and physiological experiments with the urine of such patients seem to indicate an unusual toxicity,[4] but we know nothing of the origin of this poison, nor whether it is the cause or result of the nerve disease.

It would be difficult to explain, on any toxic theory, the fact that the exophthalmos is usually greater on the side of the larger lobe of the thyroid gland.

Whether the thyroidal theory stands or falls, there is another element in the causation of Graves' disease which is so important that we ought to hold a clear conception of it in our minds. Before speaking of this, however, I shall call your attention to a few peculiarities in the history of Graves' disease, and of the patients who are afflicted with it. In the first place, it is admitted that Graves' disease often appears, and often with great suddenness, under the influence of fright or other forms of painful emotion. These are, in fact, its most common exciting causes. It is not, however, generally recognised that the symptoms may also disappear with considerable rapidity, in an acute case, provided the conditions are favourable. Dr. Gowers reports such a case, and my colleague, Dr. F. Coggeshall, has seen a very striking instance. Again, Graves' disease rarely makes its appearance except in neuropathic families. Finally, I wish to point out the very striking fact that Graves' disease may be complicated with any one or more of a variety of other disturbances of the nervous system, and finally of other organs of the body. Such accompaniments are various forms of insanity, diabetes, muscular atrophy, and even myxœdema.[1]

---

[1] Defaucomberge, cited by Horsley, *British Medical Journal*, vol. i., 1892.

[2] See Clinical Society's Report, 1886, and a paper by the writer published in the *American Journal of the Medical Sciences*, September, 1893.

[3] *Rev. de med.*, 1891, p. 1000.

[4] See for example Boinet et Silbert, *Rev. de med.*, 1892, No. 1.

The bond between these two affections may be analagous to the bond which unites Graves' disease to so many other morbid processes; that is, the existence of a neuropathic taint. Generally, to be sure, myxœdema is associated with Graves' disease, and occurs when the destructive processes which occur in the gland have reached a certain point.

There is one important principle which should be kept clearly in sight when we endeavour to trace out the ætiology of an important nervous affection like the one now before us, and this is that the symptom complexes which we see in the disease are often only exaggerations or caricatures of physiological or quasi-physiological arrangements. Thus, there is the physiological provision for rapid action of the heart to meet the needs of sudden exertion. But when this arrangement works, as one might say, too well, *i.e.*, over sensitively, we have morbid forms of palpitation. Similarly, the physiological act of attention in the presence of danger includes fixation, and perhaps even slight protrusion, of the eyeballs, and a general tension of the muscles of the body, and this often passes over into something closely resembling the symptom complex of Graves' disease. The fixed muscles begin to tremble, the heart beats come quicker, the breath comes faster, watery discharges may occur, and the whole vasomotor system is liable to be more or less paralysed.

---

[1] Sollier, *Rev. de med.*, 1893.

If all cases of Graves' disease originated in fright, we might well assume that the symptoms were due to an overstrain of medullary and other centres intimately related to the emotional centres of the brain. But even for a case not originating in fright this explanation may be made to throw light on the origin of the symptoms. It is evident that there are a set of functions, the activity of which is closely related to emotional excitement, and which form a more or less definite group. What we have to assume is that this group of correlated functions or the nervous arrangement which underlies them, form one of the weak points of the nervous system. When the nervous system as a whole suffers a severe strain, this is one of the places through which the line of fracture, so to speak, is liable to run, just as under other circumstances it runs so as to produce progressive muscular atrophy, or epilepsy, or hysteria; and when either of these latter disturbances occur in connection with Graves' disease, it is probably because the nervous system of the individual concerned has given way simultaneously in more than one direction. Usually, when the nervous system gives way at one or another point, it is possible to find some locally acting cause which made that point especially weak for the time being. This may be one of many relating causes, such as an inherited local weakness, an exceptional local strain, or, in the case of Graves' disease, local irritation of various sorts, or a temporary paralysis of the central vasomotor system. It is to the great merit of the French school to have enlightened us on these various disturbances which form what they call *the neuropathic family*, and with skill and clearness to have pointed out the relation between them. Raymond and Serieux have done this for exophthalmic goitre[1] in an interesting and valuable paper. If the lines that they draw between the different nervous affections to which they refer are too sharp, we may pardon the error for the sake of the practical value of the picture which they present. The opinion of these writers is essentially that which I have thus attempted briefly to state. They believe, that Graves' disease may be best conceived of as a disorder of the nerve arrangements in the pons and medulla, which enter into activity as a part of the emotional expression. When to this group of phenomena other nervous symptoms are added (mental symptoms, &c.), or signs of an exaggeration or giving way of other nerve activities, they consider that we have to deal, not with additional symptoms of Graves' disease, but with a series of more or less independent affections, co-existing, because all are similarly expressions of one morbid influence, namely the neuropathic state. These writers quote Peter[1] as pointing out a resemblance between the symptoms of Graves' disease and those of strong emotional excitement. But so far as the priority of conception is concerned, this view was advanced by an English writer a number of years ago (reference now unknown to me). In this connection I refer also to Darwin's work on emotion in animals. For that matter the idea is so striking that it has doubtless suggested itself to many minds independently. It is certainly a merit of the French conception of the tendency of these neurotic manifestations to show themselves coincidently that it helps us to understand the diversity of the clinical pictures presented by different patients suffering with Graves' disease. The idea would be that as the nervous system breaks down in one direction, this very fact is liable to render it more prone to break down in others on account of the excesses and excitations to which the first sickness gives rise.

---

[1] *Rev. de. med.*, 1892, No. 12.

There is one other mode of view of the pathogenesis of Graves' disease to which it is important that a few words should be given. This is the conception of it as due in part to excitation of the nerves coming from the vagus and sympathetic, which ramify so plentifully in and around the thyroid gland. This view is ably presented by Wette[1] on the basis of a large experience with the surgical treatment of goitre of all sorts. A somewhat similar position is taken by Mueller.[2] Both these writers believe that the nervous symptoms which are so common as an accompaniment of ordinary goitre pass over by insensible gradation into those which are characteristic of typical Graves' disease, though Wette inclines to the view that in the latter case symptoms are superadded, which he considers to be probably due to toxic products from the affected gland. Two of the cases of ordinary goitre reported by Wette are especially important, because they indicate how ordinary goitre is liable to affect the heart independently of the symptoms of Graves' disease. These are cases, namely, where the heart's action seemed to have been *slowed* by the irritations starting from the goitre, and we can imagine that this might happen often were it not that, as he says, the inhibitory arrangements on which this slowing depends are always the first to suffer under over-stimulation.

---

[1] *Bull. med*, April 23, 1890.

It is, indeed, improbable that the thyroid gland, with its rich nerve supply, should swell and pulsate and become filled with masses of new connective tissue without giving rise to nerve excitations, especially in view of the fact, to which I have already alluded, that irritations in the nasal cavity seem to act as the source of so many reflex symptoms, among which are occasionally exophthalmos and perhaps other of the signs of Graves' disease.

To sum the whole matter up, we have two questions to bear in mind as regards the ætiology of Graves' disease: First, what is the underlying predisposing cause? Second, what is the mechanism by which this cause acts? The underlying cause I believe to be an undue excitement of the quasi-physiological arrangement called into play frequently as a part of emotional excitement. This arrangement represents the channel into which emotional excitement naturally pours itself after rising to a certain height, and its anatomical centre is in the medulla oblongata, and related parts.

---

[1] *Arch. für Klin. Chir.*, p. 44, 1892.
[2] *Deut. Arch. für Klin. Med.*, 1898.

As regards the modes in which this nerve arrangement underlying the symptoms of Graves' disease is actually disturbed, they may be many, and among them there seems it may be that poisoning by the thyroid secretion is one active agent. This is, however, a matter of some doubt, because we have no right to assume that if the symptoms of Graves' disease are habitually due to the toxic action of the thyroid secretion, they are still capable of being excited in the same form without the aid of that secretion, and if we can get, as it appears, the greater part of these symptoms under quasi-physiological conditions, then it would seem unnecessary and somewhat gratuitous to invoke the thyroid secretion as an habitual cause of them in disease. Neverthless, it would appear that the physiological action of the thyroid extracts does excite a series of symptoms somewhat resembling those of the affection with which we are dealing.

[The exhaustive, historical and analytical monograph on Basedow's disease, by Leopold Hirsch (*Sammlung Klinischen Vorträge,* 1894) came to my notice after this paper was prepared, so that I have not attempted to utilise it fully. The author's careful analysis makes it clear that the time is not ripe for accepting any exclusive theory of the causation of the disease before us.]

As regards the *treatment* of Graves' disease, the following facts are to be borne carefully in mind: First, in the early stages of the disease it is often possible to effect an improvement or cure by the removal of the exciting cause, combined with the use of such influences as will secure the most absolute physiological rest to the nervous system. Occasionally chlorosis seems to act as such an exciting and removable cause (Robertson); at other times the irritation of some certain nerves; while in other cases the only obvious factor has been a strong emotional excitement which needs to be counteracted by profound quiet, combined with tonic treatment.

Second, one cannot review the history of the therapeutics of this affection carefully without becoming convinced that a long step toward recovery is made if one can bring to an end any one of the series of partial causes which are tending to maintain the disease. It is on this principle that we can explain the beneficial effect of the treatment of the nasal cavity, the removal of the thyroid gland, and the measures specially designed to quiet the action of the heart. The fact that by partial thyroidectomy we are able to remove a certain amount of secreting tissue of the gland, does not, I think, at all prove that the thyroid disease was the principal or only cause of the affection. What we probably accomplish in that way is the removal of a part of the excitations which combine to keep all the unstable nerve centres in an irritable state.

The records of the result of operation on the thyroid seem very favourable so far as they have been reported. Out of more than fifty cases there have been three or four deaths, and in almost all the rest a greater or less degree of improvement has resulted, frequently a substantial cure. Nevertheless, the operation should count as a dangerous one, especially in cases of weak heart. In two cases observed by myself,[1] however, the results have not been so favourable as I had been led to hope. One patient died at the end of a week, though apparently doing well. The other patient is better than before the operation, though by no means well. The thyroid at first diminished in size, but is now increasing again. I have also seen a case where a relapse occurred two years after the operation.

It happens not infrequently that an operation is followed by paresis or paralysis of one recurrent laryngeal nerve. This happened in my first case, and the paralysis has not passed away, though the voice has returned. An operation has been devised to prevent this, which consists in leaving the back part of the gland with its attachment, and removing the anterior half (Miculicz).

It is very desirable in making further studies of the effects of the operation that surgeons should pay special attention to the symptoms of the first few days after the operation, in order to test the correctness of the theory which I have advanced that a patient suffers from thyroid poisoning during that period.

---

[1] *New York Journal of Nervous and Mental Diseases*, December, 1893, and May, 1894.

The indications for and against surgical operation have been very recently, though briefly, discussed by Rehn.[1]

He thinks that operative treatment of the goitre in one or another way is the treatment *par excellence* for Graves' disease, but that if thyroidectomy is undertaken in advanced stages of the disease, or when the heart is excessively weak and irritable, it is attended with great danger. Two of his patients died from cardiac failure shortly after the operation, and it is noteworthy that in one of these cases the operation consisted only in ligaturing the thyroid arteries, so that there is no reason to accuse thyroid poisoning as having been the cause. Another patient died of pneumonia, and a fourth died on the operating table from the effects of tying the stump of the gland to check hæmorrhage. Rehn has seen benefit, in light cases and in early stages of the disease, from injections of iodoform, and recommends ligature of the arteries for very vascular goitres.

As a rule, however, he thinks resection is the best method, and that it should not be delayed until the patient is *in extremis*. Personally, I do not feel that the last word has been said with regard to the dangers of the operation or as to the means of anticipating or avoiding them.

As regards the drugs which have proved of benefit, the two most important are probably iron and belladonna. It has been suggested that belladonna acts by diminishing the secretions from the diseased thyroid gland, but it is also well known to quiet the heart in certain conditions independently of the other mode of action. I have not seen much benefit from its use. Picrotoxin was recommended by Bartholow, but Robertson tried it without any effect. Ergot occasionally seems to be useful, and iodide of mercury ointment, or some other form of iodine ointment, seems to act beneficially in reducing the size of the enlarged thyroid gland. Mental and physical rest are, however, besides thyroidectomy and the treatment of the chlorotic and anæmic conditions, undoubtedly the most effective.

---

[1] *Loc. cit.*

A patient of mine who had been severely ill for many years began to improve the moment when she commenced to lie in the open air many hours daily, and this practice she continued with benefit both winter and summer.[1] Eventually the thyroid, which had never been more than moderately large, practically disappeared, the heart's action became almost normal, and the exophthalmos much less, so that the patient has returned to moderately active life.

---

[1] See also, J. Madison Taylor, *Medical News*, December, 1893.

PERIPHERAL NEURITIS IN EXOPHTHALMIC GOITRE.

BY ARTHUR MAUDE.

In the paper I read at the opening meeting of the London Medical Society in October, I endeavoured to produce evidence, purely clinical, that peripheral neuritis was a common epiphenomenon in exophthalmic goitre. In the discussion which followed, the only notice which was taken of what was at any rate a novel point, was a direct statement from a well-known neurologist that he did not see any reason for supposing that neuritis was ever present. May I, therefore, ask to submit my views to a more highly trained audience of neurologists in these pages ?

I was at the time quite unaware of Professor Greenfield's late researches in the microscopical anatomy of exophthalmic goitre;[1] and it is premature to criticise them in detail, as they are still incomplete. I pointed out, however, that no examinations, after modern methods, have yet been made of the peripheral nerve trunks; and I still look to such examination to show changes comparable to the so-called multiple neuritis produced by various toxic agents.

The symptoms I ground my supposition on are:—

(1) The frequent occurrence of "cramp." Dr. Hector Mackenzie,[2] who first drew attention to it, found it present to some degree in thirteen out of fifteen instances, a proportion I can verify from my own experience. These painful contractions often assume the form of veritable "tetany," both of hands and feet. It is needless, since the publication

---

[1] "Bradshaw Lecture," 1893.

[2] *Lancet*, September 13, 1890.

of Ross and Bury's work,[1] to insist that muscular spasm of this form is due to peripheral irritation. As to the nature of this irritation two views present themselves.

(*a*) Tetany commonly follows removal of the thyroid, and often the injection of mucin into the circulation of animals (cats: Wagner and Hammerschlag).

It is possible, then, that cramps are in these cases the result of late, excessive disturbance of the thyroid, by which some poison, perhaps mucin, remains in excess in the circulation. The cases in which I have found cramp and tetany present have not all presented large goitres. But this proves nothing, for great functional disturbance may exist without great external increase of the thyroid.

(*b*) Tetany is frequently associated with disturbances of the alimentary canal, from the formation of a direct toxin, which may be separated from the digestive fluids. Precisely similar disturbances of alimentation are prevalent in Graves' disease. In the few cases in which I have been able to follow out this point, cramps were always associated with gastro-intestinal troubles.

(2) Ordinary sensibility is nearly always increased: "Hyperæsthesia is as universal in the sensory as tremor in the motor nervous mechanism" (Grainger Stewart[3]).

(3) Symmetrical paresis of the legs is the rule. It rarely amounts to complete paraplegia, and is never associated with rectal or cystic paralysis.

(4) The patellar reflexes are nearly always lessened or entirely abolished (Marie): this is my own experience also. On the other hand, Grainger Stewart states that the myotatic irritability varies much, that "the knee-jerks may be sometimes increased, and ankle clonus even be present, more especially in the cases manifesting a high degree of nervous excitability, but on the other hand, the knee-jerk may be entirely absent."

(5) I find varying degrees of numbness, tingling and pains in the peripheral nerves of the legs, a common cause of complaint.

---

[1] "Peripheral Neuritis," Ross and Bury, p. 398, 1893.

[2] *Edinburgh Hospital Reports.*

(6) I would suggest that those forms of localised œdema evidently of neuropathic origin, so frequent in Graves' disease,[1] are, in fact, manifestations of peripheral neuritis; comparable as they are to the conditions described by Dr. Bristowe[2] as hysterical neuritis, in which œdema was occasionally a prominent feature. The comparison is also striking between these localised œdemas and those also seen in beri-beri, which is clearly a "peripheral neuritis."

We have here, I think, all the clinical attributes of peripheral neuritis in an early stage, but in that stage it seems to stop. I have seen no cases, in record or in my own practice, which point to advanced neuritis. And as this arrest is also a feature of the neuritis of diabetes, I have examined all my cases carefully for glycosuria, with negative results. On the other hand, so many of these patients present gastro-intestinal disturbances that it is possible that the irritating poison may be of alimentary origin; while it may be some substance formed in the thyroid, or which should have been eliminated there.

Dr. Judson Bury informs me (August 29, 1893) that he remembers one case in which in addition to the ordinary tumour there was the most marked fibrillation of the interosseous muscles of the hand. This he adduces, quite correctly, as evidence of peripheral neuritis. He kindly adds: "I quite agree with you as to the probable association of peripheral neuritis and Graves' disease."

The point is, perhaps, worth following out by neurologists, both at the bedside and by microscopical examination."

---

[1] Maude, *Practitioner*, December, 1891.

[2] *British Medical Journal*, 1892, ii., p. 1098.

A CLASSIFICATION OF REFLEX ACTIONS.

BY D. FRASER HARRIS, B.SC. (LOND.), M.B., C.M. (GLASGOW).

THE accompanying classification is an attempt to exhibit in one view a scheme of the various groups of the reflex actions of the animal body arranged in ascending degrees of psychological complexity (*vide* column B.)

Both physiological and pathological reflexes are noted, though it was possible to quote only one or two examples of each subgroup out of a very large number.

The scheme aims at providing a more extensive and at the same time more definite terminology than is at present in use, whereby it would be possible to refer, with considerable precision, any given reflex to its appropriate genus or species.

The classification is more an indication of the lines on which such could be carried out than any final or rigid presentation of the question. It has however, I think, a certain naturalness and symmetry in its construction.

A terminology that simultaneously recognises the psychological and physiological aspects of the same action has its advantages.

We might include under "excito-motor," all those obviously simple or "low" reflexes which (*a*) do not require consciousness even for their existence; (*b*) do not affect it (*i.e.*, the majority of them are outside its "pale"); and (*c*) are not able to be voluntarily controlled (or "inhibited").

They are, for the most part, the actions, movements and processes of "vegetative" or "organic" life.

If, however, such a reflex affect consciousness, or be in any measure, voluntarily controlled, then it rises *psychologically* into a higher category, while its physiological nature has undergone no change.

Thus euperistalsis, conforming to *a*, *b* and *c*, is merely excito-motor; but when consciousness becomes affected as in dysperistalsis (colic), it is an algio-motor reflex—psychically higher. Again the unconscious micturition in a sleeping child is excito-muscular, whereas healthy adult micturition is sensori-muscular, consciousness being affected, and the reflex (within limits) being capable of voluntary control—physiologically they are quite similar so far as the mechanism of the act is concerned. Intermediate in psychical value between mere stimulations and the sensori-motor group are the algio-motor reflexes, for which consciousness at least of pain is a *sine quâ non.*

Under group 3, the sensori-motor, are included all those reflexes whose afferent currents have originated in a true *sense*-organ, there having existed a perception (to be strictly accurate) between the reception of the afferent current and the emission of the efferent. Now this, like most *natural* groups, has no rigid confines, for, in the first place, if the sensation be painful, the pain may be *the* predominating mental state, and the subsequent reflex be an algio-motor ; while, on the higher side, it merges into group 4 or 5, both emotions and ideas being pre-eminently stirred up by sensations *pre*sented (*re*-presented sensations being " ideas " themselves).

But when an emotion (however produced) is the *characteristic* of the mental state *causally* preceding a reflex, that reflex is emotio-motor, the emotion being *the* intermediary between the afferent and the efferent currents. Almost no state is purely perceptive, emotional or ideational, yet it may be one of these *characteristically*, and *as such* gives rise to *its* reflex.

The ideo-motor group includes all reflexes which are not the result of either perception or emotion; in some sense " residual phenomena " among reflexes.

The " ideas " may, of course, be perceptions or emotions *re*presented.

It would be under the heading " ideo-muscular " that one would classify " volitions " if disposed to do so—a proceeding very acceptable to certain advanced psychological physiologists. And yet, to call all voluntary acts reflex, seems to lose sight of certain most radical distinctions between sets of actions, most notably between a reflex act (as we all understand it), and what one might call the corresponding voluntary *imitation* of it, *e.g.*, winking from a strong light is a sensori-muscular reflex; winking *voluntarily* is *psychologically* a process of a totally different kind.

The notion of a "reflex," as a thing done in "spite of a person," vanishes. Again, laughing "in spite of yourself," is an emotio-muscular reflex (afferent currents being from ear or eye or both), whereas voluntary laughing, *i.e.*, imitation of laughing, is, on the face of it, a very different act. Of course, many reflexes can be controlled by the will, but to identify an action which is habitually uncontrolled by the will with one which must be both originated and controlled by the will seems confusing.

The will naturally suggests "reflex inhibition." This is largely, but by no means entirely, voluntary.

I suggest that a reflex action (as ordinarily understood) be designated + (positive), and that when "inhibited" or prevented from taking place (either by the restraining influence of a higher centre upon the special "reflex" centre in question, or by the advent of an impulse to the lower centre which prevents that discharge) it be called a negative (—) reflex. Under this last category might be included reflex inhibitions of acts other than reflex themselves.

Thus the will notoriously both inhibits reflexes (as in the postponement of adult defæcation) and reflexly inhibits; but a powerful "idea" can as efficiently reflexly inhibit, as when a sudden thought renders a person speechless for the time being (*negative* ideo-muscular reflex).

It is almost superfluous to add that an emotion can inhibit reflex action, as when "fear" causes dry mouth (*negative* emotio-glandular reflex), or when "joy" produces syncope (in this case a term is already in use—cardio-inhibitory reflex).

Lastly, pain can reflexly inhibit, as in syncope from "shock" from operation (negative algio-muscular reflex on cardiac muscle).

As to "vaso-motor" reflexes, if there be but *one* centre "vaso-motor," whose function it is to maintain general vascular tonus, then active (arterial) hyperæmia, due to inhibition of the centre, would be a negative excito-vascular reflex ("vaso-inhibitory" of authors), while pallor would be the positive excito-vascular reflex ("vaso-constrictor"). If there be *two* centres, one for vaso-constriction, Vc., and one for vaso-dilatation, Vd., then, *e.g.*, blushing from present emotion would be either: A + emotio-vascular reflex through Vd., or A — emotio-vascular reflex through Vc.; and similarly, emotional blanching would be either: A + emotio-vascular reflex through Vc., or A — emotio-vascular reflex through Vd.

A word as to psychico-metabolic reflexes. Of course, all vital action is "metabolic," but one can distinguish a kind of reflex concerned, not with definite glands or vessels, but having its manifestations through a wide spatial distribution.

We may, as before, have metabolism stimulated or depressed, and thus a + psychico-metabolic reflex might be more shortly named psychico-anabolic, while a — psychico-metabolic reflex would be a psychico-katabolic reflex.

Thus, the hair turning white through emotion would be a negative emotio-metabolic reflex.

The emotional state has acted "*reflexly*" on centres trophic to the nerves influencing the formation of pigment over a considerable area of skin.

It would be under the category "*ideo*-metabolic" that we would place those well-authenticated cases of hallucinations causing or curing disease. Thus there is much mystery and romance dispelled when instead of "miraculous cure by faith-healing" we write "positive ideo-metabolic (systemic) reflex!" Of course, metabolic reflexes might exist in other systems than the dermal.

A final word on reflexes. It will be very evident that "expression of the emotions" is largely reflex, while any imitation of an emotion—simulation of a particular expression—is a different thing psychologically; it is voluntary to begin with. Moreover, expression of many emotions is eminently under control: self-command does not so much consist in not experiencing certain emotions, as in being habitually able to "inhibit" or control their manifestations.

A CLASSIFICATION OF REFLEX ACTIONS ON A PSYCHICO-PHYSIOLOGICAL BASIS.

A	B	C	D	EXAMPLES.
I. PERIPHERO-MOTOR.	1. *Excito-motor.*	Excito-muscular	Of Involuntary muscle.	Euperistalsis. Micturition in sleeping child. Early non-voluntary stages of Parturition. Defæcation and micturition in "spinal disease" and in coma. Iris reflex in partial narcosis. Hiccough. Ejaculatio seminis during sleep. Later stages of deglutition.
			Of Voluntary muscle.	Spasm in children during sleep from any irritation, *e.g.*, worms. Movements of unconscious adjustment in sleep.
		Excito-glandular.		Outpouring of gastric and intestinal juices in digestion. "Sympathetic" preparation of the mammary glands in pregnancy.
		Excito-vascular.		Pathological blushing ("flushings") from indigestion. Erectio penis (in sleep). Probably certain tropho-neuroses, such as erythema, eczema, herpes, scleroderma, urticaria, "glossy skin."
		Excito-metabolic	(Dermal).	Pigmentation of areola in pregnancy. Chloasma in disease.
	2. *Algio-motor.*	Algio-muscular.		Of *Involuntary* muscle: Dysperistalsis (colic). Vomiting from "renal colic," &c. Inhibition of heart ("shock") under operation without chloroform. Of *Voluntary muscle*: Spasm=writhing from pain.
		,, glandular.		Perspiration due to pain. Lachrymation from painful foreign body.
		,, vascular.		Blushing or pallor from agony.
		,, metabolic.		
	3. *Sensori-motor.*	Sensori-muscular	Of Involuntary muscle.	Contraction of iris in strong light. Action of tensor choroideæ in positive accommodation. Contraction of uterus when child is "put to the breast." Non-voluntary elements of adult, conscious micturition and defæcation.
			Of Voluntary muscle.	Coughing, sneezing. Winking from foreign body. Laughter and spasm from tickling. Spasm from draught of cold air in hydrophobia. Deep inspiration from cold affusion.
		Sensori-glandular.		Lachrymation in strong light. Salivation on eating sour or acid materials, or at sight of food.
		,, vascular.		Pallor in cold bath. Glow of skin in hot bath. Catarrh of mucous membranes (*e.g.*, nasal) from draught of air, &c.; probably with contemporaneous "low nerve tone." Rise of blood-pressure whilst hearing music.
		,, metabolic	(dermal).	Freckles. "Bronzing" of skin in hot climates.

II. CENTRO-MOTOR.	4. *Emotio-motor.*	Emotio-muscular.	Of Involuntary muscle.	Hyper-peristalsis with diarrhœa from various emotions, *e.g.*, fear. Progress of "labour" (dilatation of "os") inhibited by stranger coming in, or induced by fright. Cardio-inhibition ("syncope") from many emotions—"Heart standing still." Rate of heart beat accelerated by emotion. Micturition through emotion.
			Of Voluntary muscle.	Child jumping for joy, stamping feet from vexation, slamming door from rage. Assault and murder from violent emotion. Emotional alteration of the respiratory rhythm, *e.g.*, sighing.
		Emotio-glandular.		Activity of certain glands, *e.g.*, stink-glands of "skunk" through fear. Weeping from emotion or inability to do so. Inhibition of salivation from fear. "Cold sweat" of fear, and perspiration from other emotions. Lactation ("draught of milk") induced through joy. Eupepsia due to happy "frame of mind."
		Emotio-vascular.		Blushing from shame, modesty, rage, &c. Pallor from rage, fear, "nervousness," &c.
		Emotio-metabolic (dermal).		Hair turning grey from emotion.
	5. *Ideo-motor.*	Ideo-muscular.	Of Involuntary muscle.	Vomiting from *recollection* of disgusting sight or taste, or from vivid gustatory hallucination. Influence of hallucinations, &c., in the insane on the muscular tonus of viscera, *e.g.*, constipation of certain lunatics. Spasm of muscles of deglutition in hydrophobia at the *idea* of "water."
			Of Voluntary muscle.	Actions and co-ordinated movements in somnambulism, and the majority of such in the hypnotic trance. Laughing at a recollection. "Reflex speech" of certain lunatics. Certain so-called "secondarily automatic" actions, such as standing, sitting, walking, playing on piano (when completely familiar with it).
		Ideo-glandular.		Weeping at a recollection. Action of hallucinations (in the insane) on secretions. Salivation at thought of food.
		Ideo-vascular.		Blushing or blanching at a recollection.
		Ideo-metabolic.		Nails ceasing to grow in mania (dermal). Action of the mind over nourishment of tissue. Mentally-induced or cured diseases (as alleged). Psychically-produced tropho-neuroses.

CASE OF TUMOUR OF PONS AND LEFT CRUS CEREBRI.

BY SEYMOUR J. SHARKEY, M.D., F.R.C.P.

W. B., aged 43, sailor, was admitted into St. Thomas's Hospital under my care on January 22, 1892, and died on the 25th of the following May.

His family history presented no features of importance. He had had malarial fever eight years previously, but had never had syphilis. Three years ago he fell from a height of twenty-eight feet, landing on his feet and fracturing his leg near the ankle, but not injuring his head in any way.

Three months and a half ago when in the Gulf of Mexico he began to perspire so freely that it became a subject of general remark. He got rather stupid, and used to knock against objects on turning to his right. Soon after he became deaf in the right ear, and suffered from numbness all down the right half of his body, the face and head being the last parts affected.

On admission he was found to have right hemianopsia, deafness in the right ear, and paralysis of the superior rectus in each eye, and the right pupil was larger than the left. Both pupils reacted to light and accommodation, but sluggishly. There was no optic neuritis.

The right ear was completely deaf, bone as well as air conduction being abolished.

There was not only numbness over the whole right side of the body, but tactile sensation was also diminished. Sense of temperature was likewise altered, and the only difference he noticed between hot and cold things applied to the right side, was that the former gave rise to a pricking sensation. Muscular sense was normal, and there was no loss of power, except perhaps in right hand. Taste and smell were not materially altered. Superficial and deep reflexes presented no marked changes. Memory said by patient to be defective, but it was not very obviously so. Speech was rather thick, and there was a tendency to stammering. Gait was somewhat peculiar, the right foot being raised higher than the left, and the heel striking the ground first. But this might have resulted from a partial ankylosis of ankle joint due to the old injury.

No inco-ordination or giddiness.

Profuse perspiration present, but no *visceral* disease detected.

During the first few days in hospital he suffered sometimes from bad headache, sometimes became strange in manner, and usually kept his left eye nearly closed, and the latter peculiarity remained unaltered till he died. He was, however, generally pleasant, and humorous, and much inclined to talk, but his memory got worse and some aphasia supervened.

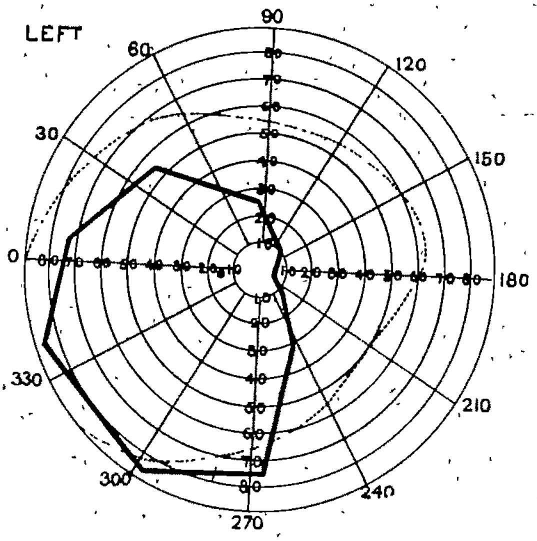
On February 1, he had forgotten the name of his last ship and captain, though he said he knew them quite well, but could not say them. When shown a candlestick and quill-pen, he was unable to name them, though he named a button correctly. When asked whether the candlestick was a watering can he became very angry, and said the question was childish and ridiculous.

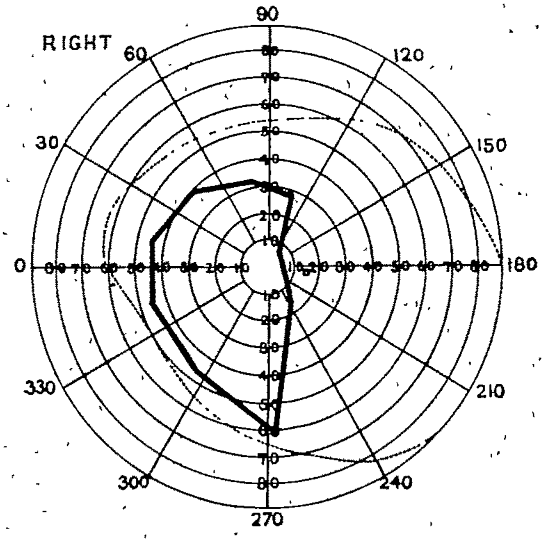
On February 12 Mr. Lawford saw him and said he could read with each eye, and with one as well as the other. He could understand what he read and could write. Loss of power in both superior recti and in inferior oblique of right eye.

On February 15 Mr. Nettleship examined him and noted that there was no movement of the eyes upward beyond the horizontal, and that downward movements were also defective, more in right than in left eye. Concomitant lateral movements full, but convergence very defective, and more so in right than in left eye. Irregular nystagmus in lateral and in upward movements. Right pupil larger than left. State of accommodation doubtful. No ptosis; but left palpebral fissure always narrower than right. As to diplopia, patient appeared to say nothing about it, and he could not with any certainty be got to see double, at any rate not in such a manner as would agree with paresis of right internal rectus.

R. reads 6J. at 14″
C. + 3 D.=1J. at 9″
L. reads 14J. at 14″
C. + 4D.=1J. at 9″
Together=6J. at 14″
C. + 3D.=1J. at 9″, but prefers to shut left eye.
R. V.=$\frac{6}{12}$: Hm. 1D.
L. V.=$\frac{6}{18}$: No Hm.

Not tried with cylinders.


LEFT
90
60
120
30
150
0
180
330
210
300
240
270
10
20
30
40
50
60
70
80


RIGHT
90
60
120
30
150
0
180
330
210
300
240
270
10
20
30
40
50
60
70
80

Fundi normal.

Loss of memory and mental power well marked. When told to sit on his locker he could not be got to understand what was wanted; nor could he say anything he wished to say. There was no muscular paralysis, but very evident anæsthesia, as he could not feel a pin stuck into his right arm. Always yawning.

On February 27 I made the following note:—

"There is distinct loss of power in the right side of face in lower part, the left side being drawn up on laughing, the right not. Tongue protruded straight. Movements of eyes good laterally, much limited in upward direction, (?) somewhat in downward direction too. Both hands grasp poorly, right less strongly than left. Legs fairly strong, but still probably not normally so; right not quite as good as left. Knee-jerks good; no clonus. Intellect very dull at times; cannot remember the names of blotting paper, pen, &c., and speech is hesitating and stuttering. So forgetful of words is he that he cannot carry on a conversation. No fits. Discs normal."

March 5.—Mental condition varies a good deal, being much better on some days than on others. Yesterday he was found sitting up in bed eating a crust of bread which he said was a pine, which he was enjoying very much; all the time he was holding his left hand below it to prevent the juice from falling on the bedclothes.

He was getting very drowsy. He could not now touch his nose with his eyes closed, or make his index fingers meet in the middle line, nor could he imitate movements made with his legs, though he was sometimes able after much thought to say which of his legs was uppermost.

On *March* 10 I made the following note:—

"Patient cannot read printed or written characters; he can make out a word or two here and there, but little more than that, and he appears to lack entirely the power of attention necessary for continued attempts. He cannot write. He very rarely names ordinary objects aright, and he does not seem to think it extraordinary if asked whether a book is an orange, apple, &c. Still he occasionally says a few sentences correctly. He is very good-humoured. Optic discs a little hazy, and streaked on internal border; (?) optic neuritis."

March 15.—Optic neuritis evident in both eyes. Attempt to take the field of vision had to be abandoned as he could not be got to understand what was wanted. His mental powers varied very markedly from day to day. For instance, on March 18 he named a bunch of keys, a watch, a knife and a pen, and said he knew other things, but that his head was "mixed." On the 21st, he could not even remember his own name. At other times, *e.g.*, March 31, he called things by wrong names; a hair brush an egg brush, a comb an egg brush, blotting paper a cork.

April 7.—Patient could not now feed himself; urine passed unconsciously sometimes; great drowsiness. Right side evidently weaker than left; clonus sometimes obtained in right foot.

April 28.—In much the same condition, at times making intelligent answers, at others unable to understand what is said to him and giving irrelevant replies; sometimes unable to pronounce words properly.

May 19.—Mentally much the same, but physically very weak; unable to sit up for any length of time.

May 24.—Very drowsy; by 5 p.m. comatose with stertorous breathing, and temperature 103·4°, pulse 132. He recovered a little and the temperature fell, but soon rose again, reaching before death 106·2°, the pulse 200, and deep coma supervening.

Autopsy.—The brain was the only organ found diseased, and after a rough examination it was put aside to harden in alcohol. On looking at the hemispheres it was seen that the left was larger than the right, but the convolutions, nerves, and vessels on both sides were normal. Where the crura cerebri emerged from the pons on the under surface of the organ the left crus was seen to be much larger than the right, measuring 1½ inch across, while the right only measured ¾ inch. The left optic tract was much flattened as it ran over the left crus cerebri.

On dividing the corpus callosum and separating the hemispheres the left crus was again seen to be very large. The corpora quadrigemina and the roof of the aqueduct of Sylvius were unrecognisable, their place being occupied by a new growth, irregular on the surface and continuous with the left crus cerebri. The whole space between the anterior and upper border of the cerebellum and the third ventricle was filled up by the tumour. The superior crura of the cerebellum could, however, be seen emerging from the tumour apparently healthy, and entering the cerebellum. The floor of the fourth ventricle viewed from above appeared to be normal. After the brain had been hardened in alcohol, sections were made.

Section 1 passed through the pons varolii vertically just beyond its junction with the medulla oblongata. The right half of the pons appeared to be healthy, but the fourth ventricle was pushed far to the right by the tumour which grew from the left half of the roof of the aqueduct of Sylvius, but the roof itself and the superior cerebellar peduncle seemed thin and healthy, as did also the greater part of the left half of the pons. The root of the tumour seemed, however, to invade the floor of the fourth ventricle.

Section 2 made vertically, ⅛ inch posterior to the emergence of the crura cerebri from the pons showed the aqueduct of Sylvius pushed far to the right, the tumour involving the roof of the aqueduct and left half of pons above the locus niger (fig. 1).

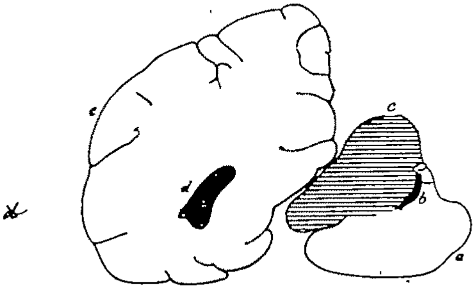


FIG. 1 (Section 2).

a. Pons varolii. *b.* Aqueduct of Sylvius. *c.* Shaded tumour. *d.* Lateral ventricle. *e.* Left hemisphere.

Section 3, made vertically through the crura cerebri about ¼ inch beyond their point of divergence, showed the right crus healthy, and the upper or dorsal part of the left entirely occupied by the tumour. The latter had obliterated the posterior part of the internal capsule, the optic thalamus, and part of the extraventricular nucleus of the corpus striatum. The hippocampal convolution was intact (fig. 2).

Section 4 just in front of corpora mammillaria. The optic thalamus, tail of caudate nucleus, and internal capsule appeared intact, the tumour appearing only in a small area of the section and involving part of the extraventricular nucleus.

Section 5 through head of caudate nucleus just behind the anterior extremity of the third ventricle. It showed the internal capsule healthy, and slight involvement of the outer and lower part of the extraventricular nucleus.

Section 6 vertically through Broca's convolution showed nothing abnormal.

The following *résumé* of the order of appearance of the symptoms helps one to realise how easy it was to come to a conclusion as to the exact position of the tumour.

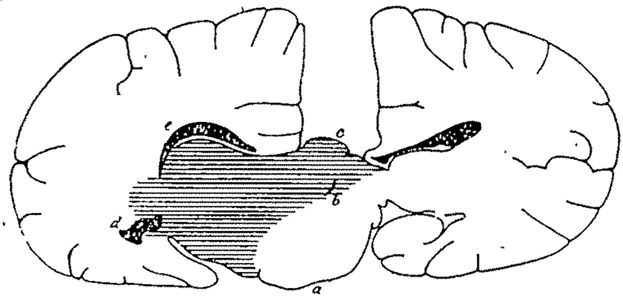


FIG. 2 (Section 3).

a. Pons varolii. *b.* Aqueduct of Sylvius. *c.* Shaded tumour. *d.* Lateral ventricle. *e.* Left hemisphere.

Three and a half months before admission:—Profuse perspiration; paralysis of superior recti of eyes (it is probable that this existed early, though only proved to exist on admission into hospital. It is not a paralysis likely to arrest the attention, and no remark was made by the patient with reference to it); hemianopia; deafness; numbness in right side.

On admission, January 22, 1892, he was found in addition to be suffering from alteration of sense of touch; alteration of sense of temperature (but no alteration of taste or smell); thickness of speech; difference in size of pupils; headache; failure of memory.

February 1.—There were found mental weakness; aphasia; utter loss of memory for words; drowsiness.

February 27.—Right facial paresis.

March 5.—Loss of taste (probably); loss of muscular sense.

March 10.—Alexia; agraphia; optic neuritis.

April 7.—Partial right hemiplegia.

May 19.—Great physical weakness.

May 24.—Coma; hyperpyrexia; death.

The paralysis of the superior recti of both sides, together with hemianopia, deafness and numbness on right side could scarcely be explained except by disease of the floor of the aqueduct of Sylvius gradually invading the left crus cerebri and internal capsule. The successive changes in the patient's condition indicated greater and greater involvement of the nerves of sense, special as well as general, and subsequently disorganisation of the more distant motor tract. It was not until late, when the tumour had reached considerable dimensions, that optic neuritis supervened.

The profuse perspiration which marked the onset of the disease, and then disappeared, is very interesting with reference to the question of the existence of sweat centres.

Another very remarkable point is the gradual abolition of mental power and of those functions which are the special attributes of the left cerebral hemisphere, owing to the cutting off of its sensory connection with the exterior, and that notwithstanding the integrity of the hemisphere itself, and that of the corpus callosum which should put it into free communication with the right hemisphere. The mental defects were very marked three months even before his death.

Critical Digest.

ON EXOPHTHALMIC GOITRE.

BY A. MAUDE.

JOFFROY[1] gives *in extenso* a series of clinical lectures delivered in 1891. He surveys carefully the whole enormous range of ascertained clinical facts, and the paper constitutes the fullest and minutest general account with which we are acquainted in any language. It is only possible to glance at some of his points. One patient, selected as a clinical example, shewed marked pulsation of the thyroid. But this is only one factor in its alteration; the purely vascular dilatation of the thyroid only exists in theory, and in books, and is never found clinically or microscopically; there is always a distinct structural change in the gland to be appreciated during life by careful manipulation; a hard consistency, usually localised and frequently nodular in distribution, these nodules being centres of inflammation or of colloid infiltration, and this conclusion is always confirmed by examinations after death if properly conducted. We do not understand M. Joffroy to deny that vascular engorgement of the thyroid does not take place frequently in healthy persons, particularly women, but that such engorgement cannot produce exophthalmic goitre. Considerable real increase may take place in the thyroid, while it remains inappreciable during life. Variations in size of the gland occur very rapidly in Graves' disease. But the point he insists upon most strongly is that the thyroid may be profoundly altered in structure and yet not increased in volume; with this we are quite in accord.

Dilatation of Arteries.—As is well known, the carotids frequently pulsate forcibly, and pulsation may be seen in the optic disc; pulsation may even be detected in the aortic arch behind the sternum. Similar pulsation is occasionally found in the abdominal aorta, but the condition is rare; Charcot described it in the earliest known case in France, and Kahler reports abdominal pulsation accompanied by paroxysms of pain. We remember that Dr. L. Guthrie[3] asserted that this feature was by no means uncommon, and he sends us a note of a quite recent observation, in which the pulsation was so violent as to be visible through the bed-clothes; it ceased abruptly at the umbilicus. The patient, a woman of 60, suffered from severe epigastric pain, and gastro-intestinal crises. Rarer still are pulsations of the arteries in the arms and legs.

Photophobia is an occasional symptom, present to a marked degree in a patient of Rendu's. Joffroy found it present, but associated it in his case with paroxysmal neuralgia of the face.

He breaks new ground on the question of *Von Grafe's symptom.* The condition has never been accurately defined even by Von Grafe. Joffroy concludes that we must understand by the term a failure of the normally even rate of movement executed by the eyeball and the upper eyelid, and this may be disturbed in two ways: (1) By a diminution of the motility of the globe itself, which thus follows the upward movement of the lid too slowly; (2) by a paresis of the lids, which consequently move more slowly than the globes in either direction.

We are convinced that neither condition is as common as some authorities (notably Von Gräfe) have asserted; and the former condition is, we think, conspicuously rare. Joffroy offers no speculations as to the seat or nature of the lesion producing these eyelid symptoms, but it may not be out of place to consider the matter briefly. It is better, we think, to group Grafe's sign and Stelwag's sign together as due to modification of the same cause. Von Gräfe himself ascribed the impaired movement to spasm in the "muscle of Muller," supplied by the sympathetic; and Gowers[3] regards it as caused by either spasm in Müller's fibres or in the levator palpebræ, probably the latter. We fear we cannot adhere to such an assumption of long continued spasm, unless due to paresis of opposing muscles.

The clinical facts are these:—

(1) Sometimes a *relative* over-action of the levator palpebræ (supplied by the third nerve), or of the unstriped muscle of the lids supplied by the sympathetic.

(2) A frequent paresis of the upper part of the facial muscles, the oculo-facial group (frontalis, corrugator supercilii and orbicularis).

(3) A frequent paresis of all the external ocular muscles.

Now the fibres which supply the oculo-facial group of muscles are derived not from the seventh nucleus, but from the oculomotor nucleus, joining the seventh nerve by way of the posterior longitudinal bundle. A slight paralytic condition in this nucleus may not be sufficient to impair the movement of the globe (though it often is sufficient), but may suffice to affect the less differentiated movements of the oculo-facial muscles, and produce in them the paresis of that group, which we know does exist, or at any rate to disturb the relation of movement in those muscles to that of the muscles of the eyeball. Such cases have no connection with the facial paralysis often observed in Graves' disease. This is due to affection of the seventh nucleus or to change in the trunk of the seventh itself. The latter was certainly the case in our own patient,[4] who had Gräfe's sign before and after the general facial paralysis, which was clearly secondary to middle ear catarrh.

Ferri[5] advances the theory that Gräfe's sign is due to mechanical shortening of the levator palpebræ, from the increased volume of blood vessels distributed in the muscle substance. This may possibly be the case when the symptom has been continuous for a long period, but the irregularity and inconstancy of the condition make it impossible that such a coarse unvarying lesion should be the usual cause.

Muscular vibration of the eyelids was found in one patient; this is common enough in hysterical patients and in persons under hypnotism, and even in ordinary debilitated subjects.

Three patients under study had marked paresis of the upper part of the face, especially of the levator palpebræ; but this paresis only affects involuntary combined movement, as in suddenly directing the eyes upwards; voluntary employment of the muscle, as in intentional wrinkling of the brows, is found unimpaired. This is the reverse of the condition often found in hysterical subjects. We may remark that Vigouroux demonstrated some years ago the frequency of impairment of electrical contractility in Graves' disease.[6]

Monoplegias.—In some cases the movements of the jaw are distinctly enfeebled. Debove and Ballet described an instance of intermittent paralysis of the tongue and soft palate.

After a brief allusion to paraplegias M. Joffroy concludes the subject of paralyses by remarking that ophthalmoplegia is most common, then paralyses of the face, the jaw, the tongue, and velum palatæ; paralysis of the arms are less common and paraplegia rarest of all. This comparison is, we think, not well founded; both from literary researches and our own personal observation paresis, and even absolute paralysis of the legs is by no means uncommon, and we think three classes of this condition may be distinguished.

(1) Cases of astasia-abasia, not of true paresis.

(2) Cases of hysterical type.

(3) Cases due to interruption of motor impulses low down in the motor tract, either spinal or peripheral. It is impossible here, however, to develop our views on this point. The paralyses of Graves' disease present a wide field of study as yet little cultivated. The difficulty is to distinguish between Graves' disease, pure and simple, and a combination with hysteria; but, M. Joffroy insists, a large proportion of "Basedowiens" do not manifest any of the ordinary features of hysteria, such as alterations in field of vision, hysterical crises, localised anæsthesias, &c.

Joffroy remarks on the abolition of patellar reflex in his patient, only to insist on the care necessary to exclude the supposition of co-existent locomotor. This, we think, is scarcely giving the symptom its true value, for Marie found it usually diminished or absent, and in investigating the point for the last few years we have found a lowered or absent reflex in nearly all severe cases.

Ophthalmoplegia Externa.—Like Ballet he distinguishes two classes of patients who present ophthalmoplegia externa: (*a*) One in which undoubted "hysterical" features are found, in whom it is impossible to assign the paralysis to either especial neurosis; (*b*) a class, less common in his belief, who cannot be considered hysterical. Paralyses of other cerebral nerves sometimes accompany ophthalmoplegia, viz., the fifth, seventh, and even the twelfth (Jendrassik).

(For bibliog. on this subject *v.* BRAIN, part lvii., vol. xv., p. 121.)

Cutaneous Rashes.—After a review of the already well-known forms of eruption or changes in pigmentation, he describes a double form of rash to us hitherto unknown. This consisted of an eruption appearing on the feet and legs resembling the later stages of the rash of measles, and mixed with this appeared a few days later a patch of small purpuric spots. This form of rash is, we say, hitherto undescribed, but by a coincidence, almost incredible, and well worth the consideration of the Psychical Research Society, on the same evening that we were reviewing this part of M. Joffroy's paper a patient of ours developed this rash suddenly.[7] She came to us two days later; there were then a few patches of purpura on the cheeks and one elbow, and over the lower part of the face and neck a large crop of raised dusky papules, resembling measles; this was accompanied by pain over the seat of distribution of the rash. The spots faded and were succeeded by a second crop of papules a week afterwards. This patient is the subject of previous goitre, and the daughter of a myxœdematous mother.[8]

Tetany.—M. Joffroy corroborates the observations of Dr. H. Mackenzie as to the frequency of spastic contractions of the hands and feet, and inclines to the opinion that they are of thyroidal origin, similar in fact to the tetany, which may supervene after removal of the thyroid.

Muscular Atrophy.—In most cases of paralysis there is no impairment of muscle nutrition. Vigouroux has described a case in which atrophy accompanied paraplegia: Dreyfus Brissac, Du Cazal, Hüber and Möbius give similar examples. Joffroy remarks that with the exception of Hüber's case, in which the occurrence of reaction of degeneration was noted in the affected arm, the cases are insufficiently reported, and there is no note as to electrical reactions.

Psychosis.—M. Joffroy's pupil, Raymond Martin,[9] collected twenty-eight cases of mental aberration in exophthalmic goitre; he lays great stress on the variety of mental changes found, and deduces the conclusion that these changes are the outcome of disturbed cerebral involution, coupled with hereditary predisposition to psychic abnormality; that there is not, in fact, any definite type of amentia, and that the cardio-vascular condition is insufficient of itself to produce change in a normal brain. We are uncertain how far this paper is inspired by M. Joffroy himself; probably to no great degree, for in a previous paragraph the professor sums up shortly the prevailing mental changes in Graves' disease, which, though they rarely amount to mania, are yet very distinct psychoses; viz., a constant restlessness, variability of temper, with alternations of excitement and depression. This alternation is very marked, and all the more marked because, though acute mania is by no means uncommon, true alternating mania (the "folie à deux" of the French) is almost unknown.

Joffroy and Achard[10] give a full account of six cases with *post-mortem* examinations of each. In five of them the thyroid was carefully examined histologically; it is only during the last few years that this has been done, and the observations in this paper are fully confirmed by the fine series of sections exhibited at his Bradshaw lecture by Professor Greenfield (1893). Joffroy's cases showed that even when the thyroid is not demonstrably enlarged during life, it was in all cases but one found enlarged after death, and in this one instance, though not enlarged, it was profoundly altered. We laid great stress last year on the fact, we believe undeniable, that all cases of Graves' disease, if properly watched, do show variations in the size of the thyroid, a turgescence similar to that of other glands during active secretion.

(2) The thyroid presents great diversity of change in all these cases. In one there was marked cystic dilatation of the vesicles. In three others the most important feature seemed to be the disappearance of the colloid material in the vesicles, or its replacement by small round cells, a condition we may regard as a catarrhal proliferation of the vesicular walls. In one instance there was a considerable formation of new adenomatous cell tissue, while the interstitial trabeculæ had undergone more or less fibroid degeneration, and this may be accompanied by œdematous swelling and infiltration of the fibrous tissue by leucocytes and interfibrous hæmorrhages.

Joffroy remarks that there is no hard and fast line of distinction between the condition of the thyroid in exophthalmic goitre and simple bronchocele, and he claims that this is not unlikely as so many cases are now on record in which apparently simple goitre has been followed by symptoms of Graves' disease. This view is quite in accord with our own, and to it we shall allude again. He raises the question whether the varieties of co-existent changes in the thyroid do not imply that the whole symptom-complex is in reality produced by an alteration in secretion in that gland dependent on one of several structural alterations.

We were, ourselves, impressed by the idea, on examining Professor Greenfield's specimens, that the various changes demonstrated (destruction of colloid, cell proliferation, adenomata, cystic and finally fibroid degeneration) were truly stages in an alteration beginning in catarrh, and ending in fibrosis or cystic degeneration, comparable to the same range of changes seen in the ovary or lung.

These six cases show a high proportion of concomitant distinct nervous conditions, viz., hysteria (1, 4 and 6), syringomyelia (3), locomotor (4), melancholia (2). The sixth case presented typical myxœdema supervening on Graves' disease. As the myxœdema advanced the goitre and exophthalmos, both previously well marked, subsided. As to the relation of the two conditions, M. Joffroy will not commit himself definitely, but he inclines to the belief that the coincidence is not accidental, but that in such cases (which are now not rarely known) an early catarrhal, or proliferation stage, in the thyroid, which is accompanied by the symptoms of Graves' disease, is gradually succeeded by progressive filroid degeneration of the gland, which, of course, produces myxœdema. We fail to see how this view can be disputed. In his myxœdematous patient the thyroid had undergone complete fibrosis, a few small cellular masses representing all that was left of the vesicles. The examination of the nervous system in all these cases was exhaustive, and disclosed these points.

Case 1.—Nervous system showed no changes.

Case 2.—The only change found was some Wallerian degeneration of the left vagus.

Case 3.—Syringomyelia.

Case 4.—Typical tabes dorsalis.—We review this case in detail later on.

Case 5.—The cervical spinal cord showed some sclerosis of Goll's columns, and columns of Burdach. There was no lesion in the restiform bodies.

Cases 1 and 4 both showed degeneration in several peripheral nerve trunks. We wish to draw attention to this as confirming our own observation that clinical symptoms of peripheral neuritis are by no means rare in Graves' disease.[11] Greenfield also observed changes scattered all over the nervous system, which (from their nature and distribution) resemble those produced in tetanus and hydrophobia, and suggest their dependence on an irritative toxic action.

O. Hezel[12] gives a full and minute account of a *post-mortem* examination of a typical case, a woman aged 45. The microscopic examination was full, and all the details of staining are given, so that we are able to say that the best methods were used. The medulla, sympathetic trunks and ganglia, the vagi, the greater number of cerebral nerves, the thyroid and muscular substance of the heart were all carefully investigated. No changes were discovered in the restiform bodies or solitary bundles. The changes in the cervical sympathetic ganglia were marked, and though limited in extent, affected portions of both sides. The main changes were :—

(1) Infiltration by round cells of the walls of the small vessels, with occasionally narrowing of their lumen.

(2) Small hæmorrhages.

(3) Nuclear proliferation of the connective tissue capsules of

the ganglion cells, often coupled with round cell infiltration in the neighbourhood.

(4) Localised degeneration of the interstitial connective tissue, often accompanied by round cell infiltration.

(5) Degenerative changes in a number of ganglion cells, exhibiting different stages of atrophy.

The conditions described here are exactly those found by Professor Greenfield in the two cases in which he has examined the sympathetic "by modern methods;" and there can be no doubt, as he says, that the sympathetic ganglia are the seat of a subacute inflammatory process. We must not, however, be too ready to accept this explanation in the face of the fact noted by Hale White[13] that the infiltration of sympathetic ganglia, apart from Graves' disease, is no uncommon feature.

The cardiac muscle was carefully examined with negative result, though there was obvious cardiac failure during life. The thyroid was considerably enlarged.

There was striking proliferation of the epithelial cells in many of the acini. The cells stand crowded together and have a cylindrical form. Many of the cells are increased in size, and the nuclei are much enlarged. The acini are frequently stuffed with epithelial cells, so that the resemblance to the catarrh of a mucous surface is at once suggested. The whole change, however, resembles not so much a simple hyperplasia as an adenomatous condition. The whole description, we should say, tallies with that of Prof. Greenfield's; the change of epithelial lining from a cubical to a columnar form, the increased proliferation similar to that seen in adenomata, and the masses of cells lying in the spaces, are described exactly as in the late Bradshaw lecture.

Professor Thomson[14] reviews eight consecutive cases in his private practice. He dismisses fright and nervous shock as a true factor in causation, being much more often an accidental event in the course of a chronic malady which seems to date its beginning. He remarks that simple irritation of the glosso-pharyngeal and superior laryngeal nerves is sometimes accompanied by general muscular tremor, and supports this by an example from his own practice, when the impaction of a fish bone caused such tremor, while its removal at once stopped the symptom. From this he argues that the tremor of strumipriva, which occurs early after the operation, is the result of direct surgical injury to the superior and recurrent laryngeal nerve branches. But we must point out, that this is ignoring the fact that tremor occurs also in the early stages of idiopathic myxœdema, and to argue thus from one instance where an alarming accident had suddenly occurred, with its attendant terror, is premature.

He then reviews, only to discredit them all, on the ground of their inconstancy, the various described lesions found in Graves' disease, and the experiments of Filehne, Seeligmüller and Bienfait. He details the progress and relapses of a patient, the subject of severe diarrhœa, in whom alternate koumiss and meat diet were pursued; the meat diet invariably produced severe relapses, ultimately fatal. He lays great stress on the frequency of diarrhœa in Graves' disease, and seeks to prove that the whole condition is due to an over-stimulation of the thyroid by some toxin, formed in the intestines from the decomposition of nitrogenous foods. Great stress is accordingly laid on the value of fermented milk, sold in New York under the name of "Matzoon." He recommends treatment by intestinal antiseptics, such as β naphthol and ichthyol. The whole paper is very suggestive and practical; and we have pointed out elsewhere our own belief that certainly some of the secondary features of this disease are due to intestinal auto-intoxication, though we are unable to follow Dr. Thomson along the whole line of his belief.

MM. Joffroy and Achard[15] discuss fully the coincidence of exophthalmic goitre and locomotor ataxy; they give an account of a patient, a woman of 49, in whom the symptoms of the two disorders progressed side by side. Ataxia was first discovered in 1879 and proptosis was then present. In 1885 the inco-ordination was marked, and muscular sense quite absent; the reflexes were abolished, there was typical swelling of the left knee, with subluxation of the joint and destruction of the internal condyle; Argyll Robertson pupils were present. The pulse rate was rapid and marked exophthalmos existed. She remained in a stationary state while under observation, and eventually died in 1891 of pulmonary tuberculosis.

A *post-mortem* examination was made, disclosing tuberculosis of both lungs. The thyroid was enlarged (weighing forty-six grammes): it showed numerous cysts, while many of the vesicles contained instead of colloid material numerous small cells; the interstitial connective tissue had undergone some fibrous increase.

The spinal cord presented the typical changes of tabes dorsalis. In the cervical region the degeneration was limited to Goll's columns. It could be traced up as far as the nucleus of the restiform bodies, but the restiform body, the solitary bundle, and other parts of the medulla were intact.

The sympathetic (right side only examined) was healthy.

The right vagus showed a few fibres in a condition of Wallerian atrophy. Various peripheral nerve trunks showed slight degenerative changes.

This is the first case combining the two conditions which has been fully examined *post-mortem*. Other cases have been reported clinically.

In this case the presence of locomotor is beyond dispute, while the examination of the thyroid, coupled with the clinical signs, also places the existence of exophthalmic goitre beyond cavil. For undoubtedly various symptoms may occur in locomotor resembling those of the other affection, such as permanent tachycardia, exophthalmos (due to paresis of ocular muscles), gastro-intestinal crises, and cutaneous anæsthesia; thyroid changes are quite unknown. Leaving out of question such cases of locomotor presenting these symptoms, there is no doubt that the spinal sclerosis and true exophthalmic goitre do co-exist. The problem is the connection between the two. The supposition has been made that the spinal degeneration has spread upwards and involved the medulla. This important case negatives such a supposition, and confirms the view that the cardio-vascular symptoms are merely by accident associated with the spinal change, as they may be associated with syringomyelia. The integrity of the restiform bodies is highly important, as opposing the theory advanced by Mendel, that those structures are the seat of the lesion in Graves' disease.

Another point of interest is the presence of tuberculosis. From our acquaintance with *post-mortem* records of Graves' disease we are disposed to say that tubercle is very rare. Greenfield raises the same question in contrasting Graves' disease and myxœdema (in which tubercle frequently appears). But we are not yet in a position to be dogmatic on this point.

Reymond[16] reports the case of a woman of 45, in whom acute symptoms of exophthalmic goitre came on very suddenly, owing to severe anxiety, and proved fatal in 14 days. The temperature ran up steadily during observation, reaching 104° on the third day. She had had continuous tremor for more than a year before she came under observation, so that this must, we think, be regarded as a "fruste" case terminating suddenly, rather than an acute case of very short duration. At the termination there was constant vomiting and dysphagia, even to liquids. *Post-mortem* mitral incompetency, which had manifested itself during life, was discovered.

The thyroid was enlarged, contained one cyst, and microscopical examination showed hyperplasia of the epithelial cells, but none of the characteristic follicular structure. We presume this may be taken to correspond to the catarrhal hyperplasia described by Greenfield and others.

The thymus was present. This is by no means uncommon. Not only is the thymus persistent in many cases of Graves' disease, but in two instances it has been at least four inches long, and in Mr. Spencer's case it was larger than the head of the normal pancreas.

The peri-bronchial and tracheal lymphatic glands were also enlarged.

F. Müller[17] reports three cases of great rapidity and acuteness, all dying two or three months after the beginning of the illness; a fourth patient was discharged apparently dying in five months. He also reports a case of ten months' duration (sub-acute) which ended very rapidly. Several of these presented bulbar symptoms (as Reymond's case with dysphagia), irregular fever, and grave psychosis, such as restlessness, somnolence, delirium, and delusions of a terrible kind. Death occurred in all from cardiac failure.

All four cases presented *post-mortem* hyperplasia of the thyroid, and enlargement of the cervical lymphatic glands, with numerous minute hæmorrhages in the glands. Three showed small recent hæmorrhages in the medulla, chiefly in the rhomboid fossa, and near the vagus nuclei. One of them had considerable degeneration of the trunks of the vagi, the recurrent laryngeal, and cardiac nerves. In one only some slight œdema of the spinal cord and bulb could be detected, and no other nervous lesion.

Müller sums up recent theories on the subject thus:

(1) The sympathetic must be definitely thrown over on clinical, physiological and pathological grounds.

(2) The medullar theory, more usually accepted, he pronounces more rational, but insufficient to explain the whole clinical picture.

(3) That the cerebrum is affected is undoubted from the frequency of psychoses, convulsions, hemiplegia and hemianopsia.

(4) That the cord also suffers is proved by the frequency of loss of patellar reflex, and the occasional co-existence of tabes, syringomyelia and Duchenne-Aran atrophy.

He concludes that the morbific agent is not limited to the medulla, but affects all the nervous centres, including probably the sympathetic. The variations and irregular progress of the disease militate against any "organic" lesion; the bulbar hæmorrhages are of no moment, being probably produced in the act of death. The inconclusive characters of his *post-mortem* are in favour of a purely "functional" disturbance of the nervous centres. Two factors in causation he considers very important: first, a neurotic predisposition either hereditary or acquired (by depression or shock); more important still, some thyroidal change. In support of the latter point he cites the now numerous cases of old-standing goitre preceding definite symptoms; the value of surgical interference: the obvious relation of myxœdema with exophthalmic goitre: the fact that the goitre is not merely hyper-vascular, but is always found to have undergone a definite hyperplasia of its tissue, and lastly, the very constant enlargement of the cervical lymphatic glands.

He deduces the supposition that the train of phenomena is the result of a functional disturbance of the thyroid, the altered secretion of which acts as a toxin, first to the lymphatic glands, and ultimately to the whole nervous system.

As evidence of our full acquiescence in these views, we may perhaps point out that an almost precisely similar summary formed the conclusion of our paper at the Medical Society in October (1893), when we had not had the advantage of seeing Professor Muller's article.

Bruhl,[18] in a long and verbose paper, loses himself in a forest of fallacies. He reports in detail the history of a woman, who died aged 61. She had at 39 developed a goitre, and came of a goitrous family, also highly neurotic, and from a goitrous district. Seventeen years after the appearance of the goitre exophthalmos was noticed, and she subsequently showed a rapid and irregular heart, and œdema of the legs. A careful *post-mortem* revealed a thyroid studded with cysts, and largely invaded with fibrous tissue, the heart hypertrophied, with some thickening of the aortic valves and the vessel itself. Nothing abnormal was discovered in the central nervous system save a small hæmorrhage in the fourth ventricle; no details are, however, given as to the histological methods employed in this examination. M. Bruhl will commit himself to no definite opinion, but is unwilling to accept this case as one of true exophthalmic goitre, and seeks to relegate it to a class of "false exophthalmic goitre," in which symptoms resembling the typical symptom-complex are presented by patients suffering from simple goitre. His grounds of argument are the district origin of his patient, her previous goitre, her age, the pathological condition of the thyroid, and the organic condition of her heart. He is, however, doubtful whether to assign her cardiac disturbance to thyroid pressure or to muscular degeneration in the heart itself.

There is no question that in goitrous districts (*e.g.*, Canton Vaud, according to Rendu) exophthalmic goitre is unknown, but this may only mean that among a mountain peasant population such a neurosis is uncommon. There is no evidence to show that, given other hereditary neuroses, Graves' disease may not be found in the midst of endemic goitre, and the number of instances is now considerable in which old-standing goitre has been followed after many years by the signs of exophthalmic goitre.[19]

We have no reliable data as to how late in life exophthalmic goitre may appear, but Russell Reynolds remarks: "Although more than half of the cases (forty-eight in number) were under thirty, yet the influence of age is reduced to *nil* if regard be paid to the number of people living in the succeeding decades."[20]

Of thirteen cases of our own, carefully watched for some years, three presented their earliest symptoms from 40 to 50 years of age, and one at over 50.

His remarks on the pathological conditions of the thyroid will not bear the light of modern researches. Professor Greenfield and (we have just seen) M. Joffroy both show that cystic and fibroid changes are the rule in old-standing Graves' disease; and the notion that the goitre is usually "pulsatile" or "vascular," is entirely erroneous.

Brühl insists that Basedow's disease may be well marked without goitre, and may even ensue after ablation of the thyroid.[21] It is, however, very easy to leave behind a fragment of thyroid or an accessory gland.

Our own reading of this instructive case is that it was a case of ordinary Graves' disease, occurring in a goitrous subject, of neurotic ancestry and personally neurotic tendency; that the preponderance of cardiac symptoms were due to the existence of ordinary cardiac and aortic degenerations, natural at such advanced age. Unfortunately, no note is made of the presence or absence of many ordinary signs, such as gastro-intestinal disturbances, though it must be remarked that no tremor was found present.

Rockwell[22] considers the therapeutics of the disease from a study of forty-five consecutive cases. Electricity is in his opinion the most valuable reagent. Convinced of the value of local galvanisation, directed to the sympathetic and præcordium, he insists that it should be accompanied by general faradisation. This corroborates the views expressed by Dr. H. Lewis Jones ("Medical Electricity," p. 312) and Max Weiss.[23] Rockwell also insists that general galvanisation should be employed also, side by side with the other methods, which we find to be the opinion of Mr. Cardew;[24] but Rockwell would employ far stronger currents than have been used formerly. For application to the sympathetic and vagus, instead of two or three milliampères (Cardew) he has employed ten or fifteen, though it is rarely possible with ordinary electrodes, to exceed this limit. For greater strengths large electrodes of clay are to be applied to the nucha (cilio-spinal centre), and the solar plexus: with such terminals he has frequently used currents of *sixty* milliampères, finding great benefit. Such currents are, of course, only admissible in the hands of trained experts, and would require caution and judgment.

Professor Putnam[25] has produced several papers on this subject lately, dealing chiefly with the question of operation on the thyroid. He gives a tabular analysis of thirty-one collected cases of operation, mostly partial or complete thyroidectomy, and attacks the two questions of the value and the danger of operation. Freiburg,[26] of Cincinnati, has collected forty-one cases, and puts the mortality at six per cent., while undoubtedly a great improvement in results has accompanied the adoption of more perfect surgical methods in the last few years. But in spite of this improvement, and the comparative restoration to health which has often ensued, there are special dangers. Patients are liable not only to sudden death within a few days of operation, but they may suffer extreme prostration, high pulse rate, laryngeal paralysis, dyspnœa, vomiting, and pulmonary complications, even when the wound is quite satisfactory.

Paresis of the larynx is very common; any pre-existing paresis contra-indicates operation, and careful examination by a laryngologist should be made beforehand. Putnam thinks that goitrous patients do not always bear anæsthetics badly, though we find[27] that many Swiss surgeons have abandoned anæsthetics in large operations on the thyroid. There seems no evidence that the nature of the thyroidal enlargement has any bearing on the result, and the forms of change in the gland are numerous. Nearly all the cases have been those of large goitre with urgent symptoms, and we yet need information as to the effect which is produced by operation on small goitres in subjects with mild symptoms.

Putnam raises the question whether the dangerous symptoms are not due to rapid absorption from the divided gland of a large amount of thyroid secretion. This is by no means improbable, and the operation practised by M. Poncet (of Lyons) seems calculated to prevent this, as also to avoid injury to the recurrent laryngeal nerve.[28] This procedure to which the name "exothyropexia" is given, consists in shelling out the gland, through a median incision, with the fingers and blunt instruments, and leaving it covered by dressings, and attached by its deep connections on the surface of the neck, where it dries up and atrophies. This operation is less applicable to cystic goitre than to general enlargements.

In a monograph shortly to be issued,[29] of which we have been favoured with an advanced copy, Dr. Edward Blake reviews some of the evidence bearing on the etiology of myxœdema and the goitres. He assumes as established that exophthalmic goitre is a toxic neuritis of the medulla, extending at times to the pons and cervical sympathetic. He lays stress on the resemblance between the effects of this toxine, whatever it may be, and those produced from the absorption of pus; while he seeks to prove that a variety of conditions, such as rheumatism, exophthalmic goitre, endemic bronchocele, are all due to an organic poison akin to the nitrites in their action on the medullary vasomotor centres, and that in a large proportion of cases their toxines are derived from the slow absorption of pus, from carious teeth, or diseased genital organs (in the female). The brochure is suggestive, and the evidence is drawn from a wide range of literature, but it is ill-arranged and not fully digested. Dr. Blake fails, we think, to disprove that all cases of so-called Graves' disease show primarily some thyroidal enlargement, and that though the question is still *sub judice* the balance of evidence is in favour of a thyroidal origin for the nerve poison.

Conclusion.—It is obvious that all over Europe there is a gradual growth of the opinion that this symptom-complex is due to the production (or non-elimination) in the thyroid itself of some toxin, which acts on the whole nervous system, even to the periphery, though the brunt of its action falls on the vasomotor centres of the medulla, and some neighbouring centres. This position is adopted in Germany by Möbius, Wette and Muller, in France by Joffroy and Renaut, in England by Byrom Bramwell, George Murray and Greenfield. The question of the exact *rôle* played by the thyroid change is difficult; it is clear that all morbid changes in the thyroid are not operative to produce Graves' disease, but all the cases (in which the gland has been properly examined) show two common factors—cell proliferation, and diminution of colloid. Even so we are in the dark as to whether this morbid change is primary or secondary to some vasomotor disturbance elsewhere, and also as to whether the alteration in thyroid secretion will produce the same effect in a healthy nervous system: probably some nervous systems are more susceptible than others to the toxic influence, just as they are to alcohol and lead; in fact the susceptibility of young women to plumbism and Graves' disease is strikingly similar.

REFERENCES.

1. *Progrès Médicale*, xviii., December 23, 1893.
2. *Medical Society's Transactions*, October 16, 1893.
3. "Diseases of the Nervous System," 1st Ed., ii., p. 818.
4. Brain, lvii., p. 122.
5. *Giorno del R Accad. di Med. di Torino*, February, 1892.
6. *Progrès Méd.*, 1887, p. 818.
7. *Med. Society's Trans.*, October 16, 1893.
8. *Australian Med. Journ.*, December, 1893.
9. *Thèse de Paris*, 1890.
10. *Arch. de Méd. Experimentale et d'Anat. Pathol.*, No. 6, November 1, 1893.
11. *Med. Soc. Trans.*, *loc. cit.*
12. *Deutsche Zeitsch. f. Nervenheilkunde*, iv., p. 353.
13. *Journal of Physiology*, viii., No. 2, p. 70; and x., No. 5, p. 345.
14. *New York Med. Journ.*, June 3, 1893.
15. *Archives de Médicine Experimentale*, vol. v., No. 8, p. 404.
16. *Bull. de la Soc. Anatomique de Paris*, ser. 5, vol. vii., No. 18, p. 456.
17. *Deutsch. Arch. f. Klin. Med.*, p. 335, 1893.
18. "Des rapports du Goître simple avec la maladie de Basedow; des faux Goitres exophthalmiques," *Gaz. des Hôp.*, p. 683, 1893.
19. Lasvenes. *Thèse de Paris*, 1891, 3 cases. Maude. *Med. Soc. Trans.*, *loc. cit.*, 7 cases.
20. *Lancet*, i., 1890, p. 1055.
21. Chretien. *Thèse de Paris*, 1888.
22. *New York Med. Record*, September 30, 1893.
23. *Centralb. f. Ges. Therap.*, July, 1892.
24. *Lancet*, p. 1, ii., 1891.
25. *Journal of Mental and Nervous Disease*, December, 1893. *Boston Med and Surg. Journal*, cxxx., No. 7, p. 153. Professor Putnam has also courteously favoured us with an advanced copy of a paper to appear shortly in the *Journal of Mental and Nervous Disease.*
26. *Med. News*, August 26, 1893. Abst. *Brit. Med. Journ.* Epit., 1893, ii., p. 78.

27. See discussion at Roy. Med. and Chirurg. Soc., November 14, 1893.
28. *Gaz. des Hôp.*, p. 362, 1894.
29. "Myxœdema, Cretinism and the Goitres," 8vo, Bristol, 1894

BIBLIOGRAPHY OF SPECIAL POINTS.

(*Connection between Graves' disease and Locomotor.*)

BARIÉ. *Bull. et mém. de la Soc. Méd. des Hôp.*, December 14, 1888, p. 503.
JOFFROY. *Bull. et mém. de la Soc. Méd. des Hôp.*, December 14, 1888, p. 514.
CHARCOT. "Leçons du Mardi," ii., p. 243.
BALLET. *Bull. et mém de la Soc. Méd. des Hôp.*, 1889, p. 76.
MARIE. "Leçons sur les maladies de la moëlle," p. 287.

(*Graves' disease and Myxœdema.*)

KOVALEWSKI. *Arch. de Neurologie*, 1889, xviii., p. 427.
SOLLIER. *Rev. de Médecine*, 1891, p. 1000.
HARRIS & WRIGHT. *Lancet*, 1892, i., p. 758.
VON JACKSH. *Soc. des méd. allemands de Prague*, January, 1893.
ORD & BOWLES. *Trans. Med. Soc. of London*, October 16, 1893.
CORKHILL. *Brit. Med. Journ.*, January 7, 1893.
GOWERS. "Dis. of Nervous System," ii., p. 880.
HARTMANN. *France Méd.*, 1884, June 17, 19.

(*Persistent Thymus.*)

MÖBIUS. "Memorabilien," xxvi., 8, 1881.
HILTON FAGGE. "Medicine," i., p. 1012.
JOHNSTONE. *Journ. of Mental Sci.*, p. 521, 1884.
HALE WHITE. *Brit. Med. Journ.*, ii., 1886, p 151.
MOSLER. *Greifwald Klinik*, 1889.
LASVENES. *Thèse de Paris*, 1891.
SPENCER, W. *Patholog. Soc. Trans.*, 1891.

HYSTERIA AND NEURASTHENIA.

BY J. MITCHELL CLARKE, M.A., M.D., M.R.C.P.
Physician to the Bristol General Hospital.

(*Concluded from page* 178, *Spring Number.*)

IV.—Papers on Hysterical Disorders of the Organs of Vision.

PROFESSOR PICK[35] thinks that the exact methods of diagnosis in use in ophthalmology are just suited to solve the difficult problem, which has been called attention to by Charcot and Buzzard, of the distinction of hysterical symptoms from those of organic nervous disease. He therefore reports two observations of the kind which also show the unmistakable influence of hypnotic suggestion.

The first patient was a girl of 24, who during infancy suffered from left ptosis and inability to raise the eyelid. Vision during childhood was so defective that she could not learn to read or write, and there was loss of movement of the eyes upwards. Except for the defective vision her health and intelligence were good, and she was employed in housework. Two years before she came under the author's notice she had been treated at an eye hospital, and the diagnosis made of congenital amblyopia. V.=$\frac{2}{60}$, similar in both eyes, with nystagmus; the fundus was normal; except for ptosis and nystagmus the eyes could be freely moved. At this time she suffered from convulsive attacks and anæsthesia of undoubtedly hysterical character, the attacks being followed by hysterical delirium. After this the amblyopia was increased to complete amaurosis. The patient was a small, strongly-built, and well-nourished woman. The skull showed great abnormality in form. The teeth were irregular and bad, the left ear deformed. The eyelids were kept closed, and could only be opened by a great effort, which was accompanied by associated action of the facial muscles. When the patient sat up her head was bent backwards, and an order to bend it forwards only resulted in its being bent still further backwards; passive movement met with strong resistance. Full extension of the upper extremities at any joint could not be accomplished voluntarily or passively. The legs were extended, and in walking the feet were dragged along the ground; she walked with extended legs and short steps. There was anæsthesia over the left side of the body and on the right leg and the back of the right hand. The patient answered all questions readily and intelligently. After an attempt at producing transference by means of a magnet the loss of sensation was transferred in the *upper* part of the body from the left to the right side, and the eyelids could be raised voluntarily for 6-8 m. without producing over action in associated muscles. Repeated applications of a magnet with one pole over each eye resulted in a remarkable improvement of vision, and two months after admission she could count fingers at 210 cm. without glasses, and at 3 metres with —3D. Later she improved still further, and could read the last row of Snellen's (capitals) types. The eye movements were fully co-ordinated outwards, upwards, and downwards, and convergence was promptly effected in a good light. The contractions in the hands grew less, and the convulsive attacks ceased. In addition to amblyopia and disturbances of motion of the eyes of congenital origin, on whose pathogenesis one cannot enter further, but which, in the author's opinion, are to be considered as related to the abnormal form of the skull, there developed on the one hand amaurosis, on the other external ophthalmoplegia of hysterical character. The proof that the last symptoms were hysterical is afforded by the fact that they came on after a convulsive attack amongst other hysterical symptoms; the ophthalmoplegia showed these characters lately regarded as hysterical; and under the influence of suggestion both returned to the state in which they were before the onset of the hysterical symptoms.

On one occasion the placing before the eyes of a —3D. lens produced improvement of vision, but a similar effect was also given by a strong + glass, and also by a tinted plane glass whose power of refraction was *nil*.

The author attributes the amaurosis to the influence of suggestion, in the hysterical condition of the patient the presence of disturbances of the oculo-motor muscles being sufficient to determine the incidence of the hysterical affection in the eye.

The second case was one in which, at the autopsy, there were found areas of cerebral softening, following thrombosis of the basilar, right and left posterior, inferior cerebellar arteries, and "art. proff. cerebri," suppurative salpingitis and chronic adhesive perimetritis. There was also consolidation of the lower lobe of the left lung. During life the patient suffered, amongst other nervous symptoms, from loss of sight, and from ext. ophthalmoplegia. Six months before death, in the early stage of these disturbances of vision, she was treated by Dr. Bayes by suggestion, by means of which vision within a few days was improved (from $\frac{4}{60}$ to $\frac{1}{36}$); the patient, however, left off the treatment because she had not improved enough to be able to read.

Prof. Knies,[36] in considering the question of unilateral visual disorders, says that the occurrence of a one-sided disturbance of vision is denied by some, affirmed by others. The question turns on the point, whether a lesion of the optic paths seated centrally to the chiasma, optic tract and primary optic ganglia can produce disturbance of vision on one side? Clinical experience shows that lesions of the optic radiations, or part of the cortex concerned in vision, produce disturbances of vision, more or less homonymous in character, on both sides. In those cases in which a one-sided visual affection has a central cause, visible anatomical alterations are not present, although for other reasons a centrally seated cause is to be assumed. A type of one-sided central disturbance of vision is that so frequently met with in hysteria, for that unilateral visual disorder is met with, and not infrequently in hysteria, cannot be doubted on account of the many observations of competent observers; and it is impossible to believe that it really exists on both sides, one having been overlooked. [It is now generally accepted that although the affection of vision predominates on one side in hysteria, careful examination nearly always detects some defect in the other field, although it may be slight.] The author is of opinion that in the majority of cases of hysteria the visual disturbance is unilateral, and suggests that when it is bilateral the central disturbance may be so too. The chief features of the affection are: (1) It is not hemiopic in character. (2) Failure of sight occurs in all grades, but seldom goes on to blindness. (3) Reaction of the pupil to light is not affected in mild cases, in complete blindness it is generally lost, but may be preserved. (4) Concentric contraction of the visual fields is found, and is in nowise always proportional to the degree of disturbance of vision. (5) The failure of colour vision is generally a typical one, and corresponds to the difference between the perception of colours in a strong light by the periphery of the retina and fovea centralis. In general, the disturbance of colour vision corresponds to the degree of narrowing of the field, and to the deficiency of central acuteness of vision.

That these visual disturbances are of central origin is shown by their occurring in connection with other hysterical symptoms of undoubtedly central origin; they are susceptible to psychical influences; they quickly come and go, and are often amenable to suggestion; they may disappear or be transferred to the opposite side, by the application of plates of metal; they may be removed by altogether insufficient means, such as putting a plane glass before the eyes; and in spite of the great contraction of the visual fields, the patients have no difficulty in seeing their way about; these last conditions make the resemblance to malingering very close. The fundus oculi is normal, and no changes appear in it after these visual troubles have lasted for years. Although certain experiments on animals (Lannegrace) appear to indicate the presence of a unilateral crossed visual disturbance of cerebral origin, yet in face of the very different formation of the central visual apparatus in animals and men, caution must be exercised in arguing from one to the other. The most serious objection to the conception of a one-sided visual disturbance of cerebral origin is that there is no anatomical evidence of a second crossing of the optic tracts above the primary optic ganglia (ant. corp. quad. and corp. genic), which on this supposition would be necessary. The author thinks that the chief facts point to a position distal to the chiasma as the seat of the lesion, and that the optic nerves may be more or less compressed at the optic foramen, and so their power of conduction impaired, but not destroyed. Thus the peripheral parts of the retina, supplied by fibres which lie in the periphery of the nerve at the optic foramen, are the most affected. The nutrition of the retina does not suffer because its vessels are not affected. In the sclerosed areas of multiple sclerosis nerve-fibres are often present, which are still capable of conducting impulses, but have lost their medullary sheaths. The most likely form of compression at the optic foramen is an annular one; this might be produced by vascular dilatation at this position, and compression of the nerves not passing on to destruction of them result. This opinion is supported by one actual observation. Leber[87] has anatomically proved, in a long-standing case of hysterical visual disturbance, the presence of atrophied fibres on the periphery of the optic nerve, especially at the position of the optic foramen. He thinks, however, that such trifling anatomical changes cannot be the cause of the visual troubles. Prof. Knies admits that the retention of the light reflex, often present in complete amaurosis, militates against his theory, but thinks that this difficulty may be got over by supposing that afferent impulses can pass to the lower centres, but that the obstruction so weakens them that they cannot reach the cortex.

To explain the frequent dependence of hysterical visual disturbance on cerebral causes, the nature of the process must be vasomotor. If this cerebral vasomotor disturbance is unilateral, defects of sensation and motion on one side will also appear. It is surely no mere accident that the only nerves which are hardly ever affected with hysterical paralysis are those which do not pass through narrow canals, namely, the nerves which pass out through the sphenoidal fissure. (The existence of a form of ophthalmoplegia externa in hysteria is now beyond question; see Dr. Bristowe, "Lectures and Essays on Diseases of the Nervous System.")

The "central" character of hysterical symptoms is accounted for by the grouping of nerve fibres in the intra-cranial canals in the same order that they occupy within the brain itself.

On these grounds the author concludes that hysteria is essentially a cerebral (and spinal?) disturbance of innervation, a great part of the symptoms arising from lesions of nerves and nerve roots, which pass through narrow canals. The common exciting causes (psychical disturbances) of hysteria produce a high degree of contraction of the (cerebral?) vessels, followed by a corresponding secondary dilatation. In those cases in which there is no exciting psychical cause, there is a manifest hysterical disposition, frequently hereditary. This may possibly be due to a deficiency of the medullary sheaths of the nerve fibres, or to defective development of them. This is pure hypothesis, but the author thinks that a quantitative estimation of the medulla of nerve fibres and of the condition of the intervertebral canals might lead to some important results.

As the result of the examination of over sixty nervous individuals, Dr. Wilbrand[38] finds that a symptom complex is present in all forms of the traumatic neurosis, consisting of contraction of the visual field, loss of acuteness of central vision, weakness of accommodation, and a great number of other visual troubles. Seeing the frequency of narrowing of the visual field and of asthenopia in all forms of nervous conditions, it would be remarkable if they were found in the traumatic neurosis so seldom as some authors say. Dr. Wilbrand, from his own observations, is of opinion that contraction of the visual field is of very frequent occurrence, and attributes the statements to the contrary of other observers to defective observation.

It may escape notice, because although an objective symptom, complaints may not be made of asthenopia, or the contraction may be present in one eye only, and that one not be examined. He attaches as much diagnostic value to slight as to great contractions of the fields. The question of simulation he thinks can be easily settled, by reason of the many methods of perimetric investigation at our disposal by which simulation can be readily detected.

Drs. Frankl Hochwart, and Topolanski,[39] from observations in Nothnagel's clinic, on non-traumatic hysteria and in neurasthenia, give the following results. In hysteria without sensory disturbance, there is generally no concentric contraction of the visual field, nor sensible disturbance of colour and light perception (fourteen cases). In hysteria, with sensory troubles, perception of light and colour may be quite normal (eleven cases). Yet although the field of vision and perception of light may be normal, the power of recognising one or the other colour may be somewhat disturbed in cases of hysteria, whether sensory disturbances are present or not.

The concentric contraction of the visual field is a pretty frequent symptom in hysteria with sensory affection (ten cases), this contraction is as a rule most pronounced on the anæsthetic, but is generally present on the other side too. With the contraction of the field of vision there is often considerable disturbance of the perception of light and colour, which in many cases corresponds in degree to the contraction of the field. The size of the field and together with it the light and colour sense fluctuate with changes in sensibility.

As an isolated symptom the authors have also observed an increase in size of the visual field and true hyperæsthesia for light and colours.

In neurasthenia there is generally no concentric contraction of the visual field (thirty-one cases). Perception of light and acuteness of vision are normal. Only occasionally is there a certain amount of loss of colour-perception, generally for blue.

The conditions in traumatic hysteria and in neurasthenia were the same as in non-traumatic cases. Over one hundred cases were examined. The authors emphasise the fact that from the method of examination there could be no question of simulation. If suspicion of simulation be aroused in the investigation of the field of vision, the testing of the perception of colour and light can be used as a confirmatory test. Decided agreement in the results of all methods of investigation shows the correctness of the observation. Where there is no contraction of the field of vision in hysteria, loss of colour-perception may be present, and together with the recognition of the sensory disturbance may sometimes aid in the diagnosis. In hysteria without objective symptoms as well as in neurasthenia it may perhaps be the only symptom capable of demonstration.

Drs. Mitchell and de Schweinitz[40] have studied eight cases, presenting various degrees of hysterical hemianæsthesia and alterations in colour vision.

"It is evident from an examination of these cases that in no instance was there loss of the colour-sense, the achromatopsia of Galezowski, nor was there any disappearance of the colours in a constant order. In the two cases of complete anæsthesia there was no departure from the normal sequence of the colours; and, practically, no limitation in the area of the field of the one (Case I.), and but slight restriction in the other (Case II.).

"In the cases of partial anæsthesia, in one (Case VI.) no abnormality for blue and red existed; green was by contrast contracted, and in two others (Cases III. and IV.) there was concentric irregular contraction of the colour-fields; but in all three the normal sequence of the colours was undisturbed.

"In two cases (IV. and V.), while the colours were correctly appreciated, their natural order was changed, the blue, red, and yellow lines crossing each other in such a manner that the red field became in some meridians the greatest in extent.

"In only one case (Case VIII.) was there an approach to complete transposition of the colours so that the red became the largest field, followed by yellow, green, and blue. In none of the cases was marked amblyopia present, except in Case VII.; here the deficiency in visual acuity was due to a high grade of hypermetropic astigmatism and concomitant convergent squint.

"We have had the opportunity of studying two cases of absolute hysterical hemianæsthesia, presenting the phenomena of crossed amblyopia. In one, a woman aged 25 years, seen by the courtesy of Dr. H. C. Wood, these symptoms appeared several months after the shock of a railroad accident. There was complete right-sided hemianæsthesia and loss of the special senses on the anæsthetic side, with amblyopia in so high a degree that quantitative light-perception was absent. The vision of the left eye was reduced to one-ninth of normal; red was the only colour correctly appreciated, and there was left lateral hemianopsia.

"In the other case, examined by the kindness of Dr. G. Oram Ring, who will publish the notes in detail, precisely the same conditions obtained, except that the amblyopia was less marked upon the anæsthetic side. In both of these cases the ophthalmoscopic appearances were normal.

"If, as may fairly be supposed, these eight cases, taken at random from a large service, represent the ordinary conditions, then our conclusions as to the occurrence of achromatopsia with hemianæsthesia must be very different from those of the European observers. The patients were selected for study because they all presented greater or less degrees of loss or change of sensibility. As to this, it should be said that we cannot find any statement by the French writers, who are the original students of this subject, concerning the relative frequency of hemianæsthesia in all hysterical cases, except statements like that of Dr. Bernutz, already quoted, that it is 'one of the primordial symptoms,' or Charcot's remark in exactly the same sense, which would imply that it was present as an almost constant characteristic. Further, as to generalised anæsthesia, or, more properly, analgesia, Bernutz calls it 'infinitely rare,' and Charcot 'relatively exceptional.' The results of the ward-service as well as of the out-patient department of the Infirmary for Nervous Diseases would not allow us to make assertions which should corroborate these. These services may, we think, be considered to represent well at least the Eastern United States, as patients come from all of them to seek treatment, and our eight quoted cases are natives of several States—Pennsylvania, Wisconsin, New York, Virginia, and New Jersey. Every case differs in the *quality*, too, of the sense-changes, and some have that very curious symptom of ability to feel heat, when great, as pain, while they cannot appreciate a needle-thrust."

In a further paper by the same authors they say that in seven cases in which one or other of the varieties of anæsthesia was a marked symptom, the most usual change was a more or less complete transposition of the red and blue lines, the red being the greater in extent. In several there was also great contraction of the visual field. In one case only in which the anæsthesia was of minor character, were the form and colour fields normal in extent. In the patient who exhibited the least marked general hysterical symptoms, there was the most remarkable deviation from the normal standard of the visual field. In cases of hysterical hyperæsthesia the disturbances in the visual field are of secondary importance; the deviations from the normal being so slight and uncertain that they could not be regarded as peculiar to that condition.

In cases classified as major hysteria there was no reversal (of red and blue fields) and no anæsthesia (in two there was contraction of the visual field), whilst in the "local" hysterias, although there was no anæsthesia, there was more or less complete transposition of the red and blue lines.

In cases classified as neurasthenia, the changes in the visual fields were not very marked, with one exception, in which the fields of vision were characteristically hysterical in composition.

In the others, if they exhibited any change it was that one most often seen in anæsthesia of the retina as described by Wilbrand. In a case of spinal injury there were characteristic (hysterical) changes in the normal appreciation of colours. The authors conclude:—

(1) Achromatopsia is not present in the American cases.

(2) Reversal of the normal sequence of the colours so that the field for red is the largest usually accompanies anæsthesia, but disturbance of colour sense is not necessarily associated with anæsthesia.

(3) The field for green is relatively more, and more often contracted than the others.

(4) Colour sense alterations are more common in hysteria than in neurasthenia, but though of diagnostic importance this is not a certain distinction, as they may be absent in typical hysteria and present in neurasthenia.

(5) One case seemed to point to the possibility of an abnormally acute colour sense in the rare cases of one-sided or general hysterical hyperæsthesia.

(6) The disturbance of colour sense is not proportionate to the severity of the hysterical manifestations.

(7) The most frequent changes in the visual fields in hysteria are:—

(*a*) Simple contraction of fields for colour but not for form.

(*b*) Contraction of fields for both; the field for green the most.

(*c*) Partial or complete reversal of normal sequence in size of colour fields, the red being most commonly the largest. The colour fields may be normal in extent or contracted.

(*d*) Loss of parts of the visual field, *e.g.*, in form of hemianopsia, or paramount contraction of the field on one side, generally the anæsthestic side of the body.

V.—Papers on Hypnotism, Hysterical Somnambulism and Double Consciousness.

Dr. Azam[*a] has collected in his book a number of papers written by him on these subjects. The works of Braid drew his attention to the study of hypnotism, and he reprints here a paper on hypnotism published in 1860, and of historical interest as the first publication in France on the subject; in this paper he gives an account of his own experiments on the lines already indicated by Braid. It is difficult to realise to-day, now that hypnotism has been the subject of such wide-spread research, that it required considerable courage in 1860 to undertake such studies, as they were then only likely to bring discredit and ridicule on the observer.

The second part of the book deals with double consciousness and somnambulism, and is largely made up of an exhaustive account of the celebrated case of Fèlida X., which was first brought to the notice of the scientific world in 1876. A short abstract of this well known case will be found in Professor James's "Principles of Psychology," vol. i., pp. 380 and 384. This patient passed into a "second" state at the age of fourteen, characterized by a complete change of character. In her "normal" or "first" state her memory only extended to the periods of her life passed in the "first" state, whilst in the "secondary" state, which in later life predominated to the almost entire exclusion of the first, she remembered the events of both "first" and "second" states.

Professor James thinks, on good grounds as it seems to us after carefully reading the case, that the secondary state was really the normal, and the primary the abnormal condition in this patient.

In a paper written in 1890, Dr. Azam says that the explanation of the curious fact that some persons have two simultaneous or alternating existences is to be found in an analysis of the phenomena of sleep, and especially of somnambulism, of which forgetfulness on waking is the principal characteristic. In this state the physical and mental activities are carried on in various degree in different cases.

The third part consists of memoirs on different subjects, being chiefly taken up by an elaborate and interesting study of the nature and origin of character, both in health and in disease, in individuals and in nations, and under different conditions of the environment.

Hypnotism.—Dr. Löwenfeld writes that hypnotism in hysteria and neurasthenia acts in several ways.

(1) Production of the hypnotic state only, a state akin to natural sleep, is useful in irritable conditions of the nervous system, in insomnia, certain forms of headache, mental anxiety and perturbation.

(2) Suggestion: Very often a condition of deep sleep is not necessary for suggestions to take effect. There are also persons so amenable to suggestion that hypnosis is unnecessary; this is especially the case in children. In other persons suggestion can only be effectual during deep sleep. Suggestions must be adapted to the condition of the patient, to his mental individuality, and to the special nature of the symptoms to be removed.

(3) Method of Breuer and Freud, see p. 125.

Therapeutically suggestion is of value in enabling isolated disturbances originating in the organ of mind, the cerebral cortex, to be acted upon apart from the symptoms of disease which may be associated with them.

Hypnotism can by no means remove the general hysterical or neurasthenical condition, but can only act on special symptoms.

As to the indications for hypnotic treatment there is much difference of opinion: many would keep it as a last resource, others would employ it at once whenever a suitable case presents itself. The author prefers the use of other measures when they will give similar results to hypnosis, and generally does not make use of hypnotism in such cases on the ground that a psychical influence (non-hypnotic suggestion) on the patient may be associated with every kind of treatment—in a certain sense *is* always so associated—and that hypnotism will not effect a cure more promptly or decidedly.

Specially capable of influence through hypnosis are hysterical symptoms of the more fixed character, paralysis, anæsthesia, hyperæsthesia, contractures, and frequently recurring convulsions, and many neurasthenic symptoms, such as loss of sleep, of appetite, and uncontrollable ideas. The drawbacks to hypnosis are the uncertainty of its action, not every patient being hypnotisable; that in full hypnosis with pronounced suggestibility, therapeutic suggestions may have no effect; that the results are often temporary, patients are relieved immediately after hypnotism, but by the evening or the next morning all their troubles have returned; and that one only out of many symptoms may be influenced by it. Hypnotism is in general more useful in hysteria than in neurasthenia. The failure of suggestion is partly attributable to the fact that to be effectual its influence must last into the post-hypnotic state, into the time after hypnosis, and this demands a high degree of susceptibility to suggestion.

The author's experience leads him to refer the unfavourable effects of hypnotism largely to want of adaptive skill on the part of the hypnotiser. In the neurotic who are prejudicially affected by procedures quite harmless in the healthy, this is a fact of especial importance, and the use of hypnosis demands prudence and careful adaptation to the patient's individuality. In persons subject to hysterical attacks these may be brought on by hypnotic procedures. Just as other therapeutic measures, cold baths, massage, &c., may have bad effects on some individuals, so may hypnotism, but this mode of treatment prudently carried out by an expert, does not certainly entail more risk in itself than most of the other means of cure daily employed in nervous diseases.

Hypnotic treatment should not however be undertaken too lightly. The possession of a battery does not *ipso facto* constitute a skilled electrician, and the mere knowledge of certain methods of inducing hypnotism does not entitle anybody to put them into practice.

The full description by Charcot of the "greater hypnotism" with its distinct phases of cataleptic, lethargic and somnambulistic conditions, has not been confirmed nor observed outside the Salpêtrière by the many investigators of hypnotic phenomena. That the observers at the Salpêtrière saw what they described Dr. Löwenfeld does not doubt, but he thinks that the phenomena were excited in persons prepared for them, and easily influenced. He considers that further experience has shown that the hypnotic state is not confined to persons of hysterical dispositions.

Dr. E. Mesnet[43] reports the case of a girl aged 17 who had suffered from hip disease, and had always been delicate and anæmic, but was never troubled with nervous disorders until March, 1872, when she had rachialgia with loss of power and sensation in the legs. From being morose and uncommunicative she abruptly underwent a change in character, becoming talkative and gay. In April, after breakfast one day, she suddenly lost consciousness and fell into a state of sleep, which lasted for an hour and from which she could not be roused by any means. When she awoke she expressed great surprise and had no memory of what had passed. The attack returned regularly for four days at the same hour, and on the fifth a second one of the same kind came on after dinner. There was complete relaxation of the limbs, loss of all the senses for communication with the outside world; the eyes were closed, with flickering of the lids. When she awoke she complained of acute pains in the head and stomach, implored aid from her relatives, but could neither hear their replies nor see them, the eyelids being closed. She attempted to escape out of the room first by the door then by the window. She struggled violently with those who opposed her movements, and showed an extraordinary amount of strength, requiring three or four persons to hold her. She afterwards talked rapidly in a delirious way for a time, then fell into a sleep like that of the beginning of the attack, and after five minutes awoke, rubbed her eyes, asked what had passed, and was surprised at the disordered state of her room, remembering nothing of the attack. The next day she had two attacks at the same hours; after twenty minutes' lethargic sleep, a period of agitation with violent declamation followed, then succeeded all sorts of violent movements, of gymnastic exercises, in which she overturned everything within reach and struggled violently when resisted, then came a period of sleep of five minutes' duration, after which she awoke, unconscious of what had passed. These attacks recurred twice a day at the same hour, varying only in duration and in intensity and in the effect of impressions received during wakefulness on the phenomena of the somnambulistic states.

All kinds of treatment including hypnotism were tried without avail. By June she had become much worse, the attacks coming at the same time but being so prolonged that she was only in her normal state for fifteen hours out of the twenty-four.

On July 1 she was removed to the Hospital Saint Antoine, and there impressed with the fact that she would be cured. As a matter of fact, without any other treatment the attacks ceased on July 3, and she returned home well on the 14th.

Dr. Cullerre[44] gives an account of a similar case of hysterical somnambulism in a girl of 20. The patient was subject to attacks of somnambulism, which would come upon her at any time, but almost always at night; in them she several times walked about the village in which she lived half naked. She was of healthy appearance; there was general cutaneous analgesia, with complete hemianæsthesia on the left side; on which also marked muscular weakness, diminution of visual acuity and dyschromatopsia existed. Whilst under Dr. Cullerre's care she had twenty-eight nocturnal attacks of somnambulism, coming on at variable hours, and varying in duration and intensity from half an hour to two hours of simple delirium, the patient not leaving her bed, to violent scenes of true somnambulism with uncontrollable actions lasting several hours. The events of the day always had a great effect on the return and character of these attacks—the most violent following lively emotions, especially disagreeable ones.

After some months she passed into a very excited mental state, one of exuberant gaiety (expansive). She recovered after some months, but relapsed after a fright a month later and was re-admitted to the asylum. She suffered then from analgesia extending nearly over the whole body. In two months the somnambulistic attacks ceased, but her character changed; from being calm and reasonable she became mischievous, with accesses of maniacal exaltation; after a few weeks she had an hystero-epileptic attack, after which her disposition improved again and at the same time the fits of somnambulism returned. The patient could be hypnotised by direct means, and the hypnotic state was that of somnambulism with closed eyes; but the occlusion of the eyes was not perfect, as she was able to recognise correctly objects such as photographs of persons known to her. There was anæsthesia except of hearing, which was abnormally acute. Contrary to the rule there was not mental passiveness. She would not, as a rule, accept suggestions made to her. Left to herself, the hypnotised patient became delirious, the delirium being a replica of that observed in the attacks of natural somnambulism. The only difference between the states of provoked and spontaneous somnambulism was that in the latter the eyes sometimes remained open. Attempts to suppress the spontaneous attacks by means of suggestion failed. In this patient it was not simply a state of noctambulism, but one of somnambulistic delirium which had always the same course and characters, in part composed of fixed hallucinations always the same, in part of varying ones, changing with the nature of the daily events in her life; thus this somnambulistic state presented the same features as those of the delirium of the third period (période des attitudes passionelles) of the fully developed hysterical attack, as described by M. P. Richet, except in one respect, the complete absence of memory by the patient of the somnambulistic attack.

Attacks of Sleep (Dr. Lowenfeld).—The absence of reaction during the attack of sleep to the different kinds of sensory stimuli, including painful stimuli, does not indicate that consciousness is abolished. Several observers have noted cases in which there was loss of reaction to external impressions—so-called "Scheintodes"—yet the sense of hearing was preserved. Hence the patient may retain some memory of the occurrences during lethargy, but this memory may be very dim for the events of the attack, or fail altogether during a certain part of it. In the majority of patients no remembrance of the experiences during the attack of sleep remains on waking. During these attacks, which for the patients form a gap in their psychic life, a certain amount of mental activity is retained. As in natural sleep they change their position from lying on the back to the side, and may carry out movements of the head and arms. Thus one patient—who suffered often from chilliness—wrapped herself up on these occasions in a woollen covering, and if this was drawn away so that the upper part of the trunk and the arms were exposed, she wrapped the covering around her again. Another patient reacted to a suggestion made in her hearing whilst she was in lethargy that she should go to a concert if she awaked half an hour before the appointed time for it.

Hysterical Somnambulism.—Dr. Löwenfeld adopts Joseph Frank's division into (1) somniatio, (2) noctambulism. Several groups or varieties exist. Attacks of somnambulism may be attended by pronounced and easily recognisable mental disturbance or by such slight mental change that it can only be recognised by those who have an intimate acquaintance with the patient's normal state. As a rule the patient in his normal mental condition is unaware of all that has occurred during the period of hysterical somnambulism; only very seldom does a dim remembrance of single events of the somnambulistic state remain. The somnambulistic bears the same relation to the normal mental state that a tumour does to the rest of the body. The mental attainments and character of the patient in the two states are often different. The second or somnambulistic state may attain such a duration and importance that a doubling of the personality can properly be spoken of, a division of the mental life into two psychical existences distinct from each other.

In the majority of cases the patients are not in the full possession of their natural intellectual powers during the somnambulistic state. Prolonged somnambulistic conditions often follow convulsive seizures or attacks of sleep, and the return to the normal may be similarly brought about. Hysterical somnambulism differs from attacks of psychical epilepsy with ambulatory automatism in that in the latter no remembrance of the events that have taken place during them can ever be aroused, whereas in hysterical automatism, a return of the secondary state, which may be brought about by hypnotism, brings back with it the remembrance of past acts of the original or of other previous attacks.

In concluding a paper on "Cases of Hysteria treated by Hypnotism," Dr. S. G. Webber,[45] of Boston, remarks: "The first is a complicated case. There was a peculiar condition of the nervous system, a condition of neurasthenia, also a hysterical state. In some way, which I do not understand, the patient came into a state in which she lost her memory of recent events, and her relation to those about her were changed to such an extent that she did not recognise those whom she had seen and known only a few hours previously. Then she returned to her normal condition, and had no recollection of what had just occurred. Her brain then became a dual organ; in some way the awaking of one set of ideas and associations in one group of nerve cells seemed to inhibit the activity of the group of cells which was cognisant of the other set of ideas and associations.

"Later, when I had put her to sleep, there was the manifestation of other phenomena; there was not only the two states of brain activity, but also the power or ability of receiving suggestions when all the mental faculties seemed to be dormant, and it would have been supposed that she could not hear nor the brain receive any impression, at least so as to have a permanent effect.

"Memory, consciousness and will were perverted, or in the artificial sleep they seemed to be abolished; certain groups of nerve-cells, which are usually active during waking hours, were inhibited in their action. For a while they were placed in an enforced quiet. This was seen in the second case. I could easily put the patient to sleep; there was no cataleptic state of the muscles; she simply went to sleep, a quiet sleep such as anyone might have, with dreams or without as the case might be. She was, however, insensible to all noises, was not disturbed by the rattling of paper, opening of table drawers, the slamming of doors outside, nor by my moving about the room. As soon, however, as I spoke to her, she showed that my voice reached her brain, there was a change in the expression of her face, and if I continued to speak she soon waked up.

"It will be interesting to compare these peculiar mental conditions with what is seen in an ordinary case of delirium, in which there is a more or less complete unconsciousness of one's surroundings. I will report a very simple case which resembles hypnotism.

"Miss A. has had severe headaches, especially at the time of her catamenia, and in these is sometimes delirious.

"March 20, was called to her about seven in the evening, as she was delirious. I found her lying on her right side; the muscles of her neck and arms would occasionally twitch, and she carried her hand to her neck every now and then, and showed by slight groans and sighs that she had severe pain. She seemed to imagine herself at home, and called her mother in a rather low tone of voice. When the nurse came she told her to go for her mother. She seemed to think I was her uncle Edward. She tried to get up once or twice, and said she must go or be going. There was no violence and no loud outcry; she was simply in a quiet delirium. She did not realise where she was nor what she said, yet seemed to realise that she was in bed, and tried not to uncover herself. Under the use of remedies and massage to the head she fell asleep, awoke at a slight noise, then went to sleep again, and soon woke up in her right mind, astonished to find me by her, and asked if she had done anything out of the way.

"In this case there was an entire misapprehension of her condition and surroundings. She was as much out of touch with those about her as the hypnotic, more so in fact. Yet she retained a certain amount of receptivity; she knew that someone was near, and made known her request that her mother should come. In this patient the memory and consciousness were perverted or suspended, the will was still active, but acted in an abnormal way, because it had not correct data to guide it. Ideation was more affected. In hypnotism there was a more orderly sequence of ideas; in delirium the sequence of ideas was less regular; slighter influences served to give rise to new ideas not in harmony with those preceding. It seems to me this may be one of the differences between hypnotism and delirium; not only are memory, consciousness and will affected, ideation is irregular and dependent upon accidental influences, arising outside or perhaps within the patient. In hypnotism, when ideation is affected, it is perverted according to the will or suggestion of another.

"It would be interesting to compare these two states with dreaming. This seems to be a modification of the above conditions, the cause being within the brain itself, or often coming from without. But in dreaming there is less order; the control of the thoughts, which is not wholly lost in hypnotism, and which still is found to some extent in delirium, is very imperfect in dreaming; the logical faculty is more affected, or it may be said the memory is more at fault and does not act to regulate the sequence of ideas, so these run riot in the brain, and the different groups of nerve-cells are awakened to activity and silenced in a most unorderly manner, giving rise to the most absurd and incongruous grouping of ideas and images.

"Dreaming is likely then to be the least orderly of these abnormal activities. There are, to be sure, dreams which seem to be perfectly orderly and logical. Perhaps they are so, or it may be that they seem to have this order because the waking mind unconsciously reduces the mass of ideas to something approaching order. It would be interesting to follow the analogies in these states much further."

VI.—Papers on the Association of Hysteria with Organic Nervous Disease.

Dr. Babinski,[46] in discussing the association of hysteria with organic and other nervous diseases, says that the affections which are first and foremost, disseminated sclerosis, then tabes dorsalis, have been especially noticed as frequently associated with hysteria syringomyelia, Morvan's disease, Pott's disease, facial paralysis, the primary myopathies, neurasthenia, agoraphobia, onomatomania, chorea, and exophthalmic goitre. It is reasonable to suppose that if a slight injury or simple emotion can give rise to the appearance of hysteria, organic affections of the nervous system, with the profound changes that they entail, should have analogous effects. Such a combination of hysteria and organic disease is obviously likely to be a fruitful source of mistakes in diagnosis, and it is to failure of recognition of this combination that the opinion put forward by some persons that psychotherapeutics may modify or even cure organic disease is in part due.

Case 1.—A woman of 45, alcoholic, suffering from right hemiplegia, the arm being most affected, with rigidity of muscles and exaggeration of reflexes, as the sequel of an apoplectic attack six months previously. There was also right hemianæsthesia, with contraction of the right field of vision of the same duration. The sensory symptoms were removed by the application of a magnet.

Case 2.—A man aged 31, who had suffered from syphilis ten years before. The illness was of one year's duration, and consisted of progressive mental enfeeblement and loss of motor power. The mental state was one of complete apathy, with inaptitude for any intellectual operations. There was also a peculiar difficulty in speech, trembling of the upper lip, and slowness of reaction of the pupil to light. The upper extremities were normal, the lower could be moved freely when the patient was lying down, but he could not stand or walk without support; when he tried to do so his legs gave way under him. Sensation was deficient over the legs; the tendon reflexes were normal. Micturition and defæcation were naturally performed.

Faradisation of the lower extremities, together with a statement made to the patient that electricity would cure his paralysis, resulted in immediate improvement, and in ten days' time he could stand and walk fairly well.

The author says that this case is one of meningo-encephalitis with hysterical astasia, but he admits that the cure was not complete, and that, therefore, the paresis of the legs might be in part due to the cerebral lesion, but he considers that it was principally due to a purely dynamic or hysterical disturbance associated with it.

Case 3.—A female cook, aged 42, suffered from hysterical fits, followed by choreiform movements which lasted for a year; this occurred when she was 21 years of age. In 1892 she was knocked down by a carriage and sustained a fracture of the right petrous bone with permanent facial paralysis; two weeks later, attacks of vertigo and loss of the power of maintaining her equilibrium prevented her from standing or walking. When lying down she had no vertigo and no loss of motor power except a little feebleness in the right hand, but when sitting or standing she swayed from side to side, and the gait in walking resembled that of a drunken man. She had also hemianæsthesia, together with dulled hearing and contraction of the visual field on the right side. These motor disturbances were cured very speedily by suggestion.

Case 4.—A peripheral neuritis of mild intensity affecting a woman who had formerly had hysterical attacks. The neuritis affected chiefly the sensory fibres, and its chief signs were lightning pains, absence of tendon reflexes, and diplopia. In addition were present symptoms, which appeared to be almost exclusively due to hysteria; these were muscular enfeeblement, anæsthesia and vesical troubles, which disappeared under the influence of suggestion. The disturbances due to neuritis gradually grew better. The above-named symptoms were considered to be hysterical because the loss of power was out of proportion to the slight degree of neuritis, the electrical reactions of the muscles being normal, and on account of the influence of suggestion. The other cases included hysterical retention of urine in connection with cystitis, cured by suggestion; attacks of hysterical sleep in the course of a case of neurasthenia; and hysterical symptoms complicating endometritis.

Clinical Notes of Cases of Hysteria reported by Dr. Ghilarducci from the wards of M. Charcot.[47]—In the first, there were attacks of typical sensory epilepsy; numbness invading one half of the body, including the tongue; paralysis of the limbs, motor aphasia, and mental disturbances, chiefly loss of memory. The family and personal history of the patient pointed to tuberculosis, and at one time the diagnosis of a lesion of the cortex was made. The signs of hysteria were the occurrence of hystero-epileptic attacks and the presence of the permanent stigmata.

In the second patient, attacks resembling those of Jacksonian epilepsy from a lesion in the neighbourhood of Broca's convolution occurred, accompanied or preceded by paresis and tremor of the right arm and motor aphasia, ending in general contracture of the muscles, loss of consciousness, and biting of the tongue.

The third patient, besides well-marked symptoms of hysteria, suffered from attacks beginning with cramps in the calf of the left leg, a feeling of cold which passed rapidly over one half of the body, and was followed by tonic and chronic contractions of the two limbs most marked in the lower extremity.

In the fourth case, the symptoms took the form of localised epileptiform convulsions, resembling the evolution of cerebral syphilis. There are also accounts of a case of "hysterical apoplexy" *i.e.*, of hysterical hemiplegia with unusual characters. All these cases are reported in great detail, and the points in the differential diagnosis of each of them fully entered into.

M. Babinski,[48] in another paper on organic and hysterical contractions, observes that according to Brissaud and P. Richer hysterical contractures present the closest analogy to spastic contractions due to sclerosis of the lateral columns; in both there is muscular rigidity, together with exaggeration of the tendon reflexes, and sometimes ankle clonus. The author, however, believes that apart from any other symptoms in other parts of the body, indicating either hysterical or organic disease, the characters of an hysterical contraction are, in themselves, pathognomonic. As a point of distinction in organic hemiplegia the author lays stress on a feature of the paralysis in the upper extremity that Dr. Buzzard has previously pointed out. Dr. Buzzard[49] says: "In true hemiplegic contracture . . . you cannot, by any amount of force, straighten the whole limb at one moment. If you straighten out the fingers the wrist remains rigidly flexed. Bring the metatarsus into a line with the forearm by extending the wrist, the fingers will *ipso facto* become rigidly flexed." In hysterical contracture the forearm, hand, and fingers can be brought into the same plane. Of equal importance is the exaggeration of the tendon reflexes in organic disease. M. Babinski thinks that a manifest and unilateral exaggeration of tendon reflexes in a case of spasmodic hemiplegia indicates, as a rule, that the motor troubles are not hysterical; this holds good in a case of paraplegia, if the bilateral exaggeration of reflexes is very pronounced and accompanied by ankle clonus.

The reasons for the above statements are: (1) In a very large number of cases of spontaneous or provoked hysterical contractures in hypnotised subjects, the author has found no notable modification of the tendon reflexes. (2) The independence of hysterical contraction and of exaggeration of the tendon reflexes was well shown in a case of right hemiplegia. In this patient an hypnotic, *i.e.*, hysterical contracture could easily be provoked on the left side, and this contracture did not modify, in any way, the tendon reflexes. On the right side an hypnotic contracture could be provoked by suggestion in the lower extremity, in which the motor power was preserved relatively to the arm, but this contracture was much less intense than that evoked in the opposite limb. It was, however, impossible to modify the condition of the right upper extremity, in which there was marked paralysis, either to augment the rigidity already existing in it by hypnosis, or to produce an hysterical contracture. (3) In a case of tabes with absent knee-jerks, pronounced contracture of the lower limbs could be produced during hypnosis. Since observations, contradictory to these statements, have been made by competent observers, is it possible that two forms of hysterical contraction exist, one without and one with exaggeration of tendon reflexes? The author thinks that the last condition must be altogether exceptional, although he will not directly deny its existence; perhaps the difficulties and pitfalls which lie in the way of exact observations on the state of the reflexes may account for some of the discrepancies in the statements. He points out that exaggeration of reflexes may be apparent only, and be counterfeited by other reflex or voluntary movements, excited by the percussion of the tendons. Difficulties of diagnosis may obviously be great where hysterical is associated with contracture of organic origin, but in these cases the latter cannot be very marked.

With regard to the nature of these contractures M. Babinski remarks that relaxation is as active a change as contraction of a muscle; although certain features of these contractures correspond to those of voluntary contraction, yet the spasmodic rigidity of organic disease cannot be considered as identical with prolonged muscular contraction, for it is impossible to voluntarily produce, even transitorily, a state of muscular rigidity with the objective characters of organic rigidity (contracture). As to hysterical contracture, however, it is possible to produce, during a short time, a muscular rigidity which corresponds exactly in objective characters to hysterical contracture, and in ordinary hysterical cases a contracture may succeed a voluntary contraction and the inequality of the duration of each constitute the only difference between them. A patient, with hysterical contracture, departs from the normal in the length of time he is able to maintain a contraction, or rather in that he is unable to relax his muscles after contraction. Comparing hysterical contracture and paralysis, in the former the patient is unable to induce relaxation, in the latter contraction of the muscles affected.

Hysteria and Disseminated Sclerosis (Löwenfeld).—With regard to the diagnosis between disseminated sclerosis and the forms of hysteria which resemble it so closely, the author remarks that the hysterical tremor and movement generally continues during rest, and is not a true tremor occurring only with voluntary movement, but one that merely *increases* during movement, shows great alterations in intensity, and may suddenly disappear. In hysterical speech affections the monotonous scanning speech of disseminated sclerosis is not observed, but rather a form of stuttering. If hemiplegia is present it is characterised as hysterical by the accompanying hemianæsthesia, glosso-labial spasm, absence of facial paralysis and of the signs of descending sclerosis. The nystagmus and pallor or atrophy of optic disc, common in disseminated sclerosis, are absent in hysteria. See also Buzzard.[60]

Prof. Grasset[61] reports the case of a woman, aged 23, suffering from hysteria and disseminated sclerosis, in which the symptoms of disseminated sclerosis were nystagmus, scanning speech, characteristic tremor of the limbs and head during voluntary movement only. Exaggeration of the knee-jerks and ankle clonus. No affection of sensation, except slight diminution in the right leg. The special senses were normal except vision. As to the latter there was (1) considerable, but irregularly, concentric contraction of both visual fields—the outline differing from hysteria in its marked irregularity; (2) no dyschromatopsia; (3) atrophy of optic discs consecutive to neuritis.

Five weeks after admission the patient had two hysterical convulsive attacks, caused by a strong emotion. Besides the defective sensation in the right lower extremity, other hysterical symptoms now present were hysterogenic zones over the ovaries and under one breast, and loss of the pharyngeal reflex.

About eight months later the patient was, without warning, seized with serious bulbar symptoms and died in a short time. At the autopsy the bulb and cord only could be examined; the bulb presented a "compact sclerosis," with some more distinct points of better defined sclerosis; in the cord were several characteristic patches of sclerosis, most numerous in the cervical and lumbar enlargements.

Professor Charcot[63] relates the case of a young man, aged 31, who discovered whilst in his bath that his left lower extremity was markedly wasted, the foot and the leg being chiefly affected. His astonishment and fright was so great that he had an hysterical attack shortly after noticing this. No loss of power or affection of sensibility had occurred to lead him to think there was anything wrong with his leg. On examination, the circumference at lower third of thigh, right 38 cm., left 34 cm.; at middle of calf, right 38 cm., left 31 cm. There was some spasm of the extensors of the toes. The atrophied muscles were those of the foot, leg, thigh and buttock; they were the seat of fibrillary tremors, but there was only very slight loss of power in them, and the electrical reactions were in all normal. The patient could walk perfectly naturally. The knee-jerks were exaggerated; no ankle clonus, no alteration in the colour, temperature or sensibility of the limb. The patient came of a family prone to hysterical affections, and had shown signs of hysteria himself. It should have been said that the muscles of the right shoulder and upper arm also showed commencing atrophy, fibrillary tremors, and exaggerated tendon reflexes. Professor Charcot points out that the above symptoms with absence of change in electrical reactions in any of the affected muscles exclude lesions of the nerves, cord or brain; but rather consort with the hypothesis of purely functional or dynamic lesion. Hysteria is capable of causing such muscular atrophies with characters that simulate atrophies of spinal origin, and which follow the course usual in other hysterical manifestations appearing and disappearing with them. Facts of this kind have been recorded by Kahlkof working under Seligmuller, Babinski, Blocq, Debove and others. These hysterical muscular atrophies coincide frequently with paralysis or anæsthesia of the affected limb; they are sometimes developed in a few days, and after a variable time pass off either slowly or quickly. They are not accompanied by fibrillary contractions; recent observations, however, have shown that hysterical muscular atrophy may be attended with fibrillary tremor, and the seat of the atrophy with the exaggeration of the reflexes sometimes met with in hysteria make the resemblance to atrophy of spinal origin a strong one. These cases are, however, exceptional, and an electrical examination will settle the diagnosis, for so far the reaction of degeneration has never been found in hysterical atrophy. Such being the features of hysterical muscular atrophy, there is nothing to absolutely gainsay the dynamic or functional origin of the muscular atrophy in the above case.

VII.—Papers on the Traumatic Neurosis.

Dr. Strümpell,[38] in discussing the traumatic neuroses, says that for some years, especially since the establishment of legislation for compensation for accident, physicians have had frequent opportunities of observing patients who complain of a group of undoubtedly nervous symptoms as the result of an accident. Before the accident they were quite well, and in one group of these cases the connection between the injury and the nervous disturbances is undoubtedly clear. Whatever be the nature of the injury, the character of the illness can be estimated by the ordinary means of general and local diagnosis.

Another group of cases presents very different features. The injury has not fallen on a part where important nerve structures lie, or has not been a very severe one, so that a severe effect on any part soever of the nervous system is not probable. The kind of pains and nervous disturbances suffered by the patients are not of the kind that occur in connection with grave nerve-lesions. The symptoms are many of them of a general nature, spread beyond the injured part, and certain symptoms are always absent which are met with in organic nervous affections. The characters of the cases in this group are distinct from those produced by grave injuries of the nervous system, and correspond to those found in functional nervous affections. As it is the custom to call these functional affections "neuroses," this group of cases has been called "traumatic neurosis," in order on the one hand to emphasise the connection with injury, and on the other the functional nature of the disturbance.

The term *traumatic neurosis* was never intended to be used to indicate a special and fixed disease, but rather as a short designation provisionally acceptable for a group of morbid conditions, whose clinical symptoms and pathogenesis must first be established by special investigations. The use of the expression the *traumatic neuroses* should have indicated that the conception of a special traumatic neurosis was in no wise permissible. The group of symptoms are in fact not by any means always the same. With the view of a provisional sifting and grouping of the cases, the author has separated them into (1) cases with clearly local nervous symptoms (pains, paralyses, contractures, anæsthesias, &c.), (2) cases with undoubted general symptoms of disturbance of central nervous system, without in any way intending to make "local traumatic neurosis" a special disease. He then briefly describes the form of disease which is commonly meant when a "traumatic neurosis" is spoken of.

The patient has suffered from the effects of a fall or blow for a longer or shorter time, and although the immediate effects of the injury have long passed away, he has never regained his former health. He attributes his altered physical condition directly to the injury. Though a blow on the head or chest may cause pains in the head or chest respectively, yet the pains are never entirely localised, and correspond to no special nerve distribution. With the pains is a corresponding loss of motor power, which renders all active bodily work impossible. Together with loss of physical strength, there is incapacity for mental exertion, insomnia, loss of appetite, palpitation, sense of pressure in the head, &c. "The more the patient is questioned as to special symptoms, the more complaints are obtained in answer." The objective signs of disease are, in comparison with the subjective, insignificant. Only after close examination of all the nervous functions does one hit upon those symptoms which Oppenheim first laid stress upon as "the objective signs of the traumatic neurosis," and the word "objective" must be taken *cum grano salis*. The chief objective symptoms are the various anæsthesias and hyperæsthesias of the skin, affections of the special senses, contraction of the visual fields, &c. They are real and important symptoms, but depend on the "state of consciousness" of the patient, and their significance must on that account be altogether different from the independent and objective signs of organic nerve disease. The skilful physician will be able to recognise the real origin of these symptoms from the character and extent of the affections of sensation and of the special senses. The contradictory character, want of uniformity, and variability of the symptoms is only striking to the unlearned. And yet it is quite intelligible that the statements of different physicians over the same morbid state should be at variance. For the nature of the clinical investigation has a great influence on symptoms of psychic origin. For instance, he who constantly searches for contraction of the visual fields will be more likely to meet with it frequently than the man who doubts the existence of this symptom. Many so-called *objective laws* of hysterical phenomena rest on the operation by suggestion of the method of investigation.

Whether the term *traumatic neurosis* is retained or abandoned, and replaced by the names *traumatic hysteria* and *traumatic neurasthenia*, is indifferent so long as one holds fast to a sharply defined definition of what is understood by this name. Practically it appears reasonable to provisionally retain the name traumatic neurosis as a collective term, whilst allowing that it does not exist as a specific disease.

Dr. Strümpell raises some specially important points in the differential diagnosis. Fr. Schultze has pointed out that many patients refer their troubles to an injury, whilst they are suffering from a general morbid process such as arterio-sclerosis or alcoholism. Further, the decision that a material nerve injury of some kind or other has not taken place in some of the cases of predominant local symptoms is by no means an easy one, especially in the presence of fractures, deep wounds, &c.

Perhaps certain reported cases of *traumatic neurosis* in which such anatomical changes as *optic atrophy*, *fixity of pupil* were observed, must be referred to chronic organic changes produced by the injury (commotionstorung). The writer has observed neuroses which went on to a well-marked progressive paralysis. He has seldom observed intentional simulation, and points out that simulation in traumatic neurosis is not at all easy. How should a simple workman, totally ignorant of the observations of the Salpêtrière school, be able to simulate loss of smell and taste, and hemianæsthesia? The constancy of the symptoms would imply a special pathological process. In diagnosis the author would put more weight on the *tout-ensemble* of the disease, and general mental impression than on single symptoms, and thinks that isolated persistent objective symptoms, even when found, have only a relative value. But it must never be forgotten that all symptoms of disease dependent upon psychical conditions follow quite different rules from those which have an anatomical basis.

If the reality of the disease is to be put in doubt on account of the instability of certain symptoms, and of the exaggerations of the patient, then half one's private patients must be simulators for the same reason.

However clear the diagnosis of single cases may be clinically, their forensic consideration is generally difficult. How far the insurance companies must be liable, how far allowance must be made for the subjective psychical factor which plays such a great *rolè* in the causation of the neurosis is questionable. Treatment should be taken in hand at once, as the bad prognosis and want of response to treatment in this disorder results partly from the fact that the patients go for months or years from one doctor to another, from one court to another, till the symptoms of their malady become fixed and graven in their innermost being. The rule for treatment is "Principiis obsta"!

Dr. Ch. Vibert,[64] as medical expert to the tribunal of the Seine, has had the opportunity of making numerous observations on the results of severe accidents. He has been able to follow up a certain number of cases for several years, and has learnt from them that the disorders which develop consecutively on an accident may, after an interval of apparent good health, go on developing in intensity even to a fatal termination.

Five persons in the Charenton railway accident, who had no external wound nor fracture, had punctiform hæmorrhages over the whole or half of the upper part of the body, together with deep congestion of the face, and sub-conjunctival hæmorrhages. Similar appearances were found in persons with fractured skull, or extensive wounds of the head, and the author is inclined to attribute them to lesion of the nervous centres, probably the bulb, but has no direct *post-mortem* evidence of this. The lower extremities are much more frequently damaged than the upper in railway accidents. Insomnia, muscular tremor, a highly-emotional state, or one of hebetude, stupor, or a semi-conscious condition are the nervous conditions which most frequently directly follow the accident, and as a rule disappear in a fortnight's time. This nervous shock plays no part in the development of the grave cerebral disorders which follow an accident unless the brain itself has been injured by concussion or commotion, except that sometimes excessive emotion causes more profound and lasting troubles than those mentioned above.

The most common general symptoms are loss of appetite, and various digestive disturbances, asthenia, depression, loss of flesh, anæmia, and profuse sweating on any exertion.

In the case of a diabetic who sustained contusions of the head, and a severe wound of the leg in a railway accident, the glycosuria was not aggravated.

Cases of direct or indirect injury to the lungs and stomach are given in detail.

Cerebral troubles occasionally appear after an interval of some weeks of health has succeeded the disappearance of the first symptoms of shock, but in the majority of cases they come on immediately after the accident. These disorders are *intellectual*, as loss of memory, loss of the power of attention, melancholy, apathy, indifference, irascibility, emotional excess, headache, often vertigo; all aggravated by intellectual work, emotion, and especially by alcohol.

Disorders of special senses.—Ears: Tinnitus aurium, sometimes deafness, inability to bear any noise. Eyes: Photophobia, paresis of accommodation. Taste and smell are rarely affected.

General sensation.—Subjective sensations in the limbs; the author has not observed anæsthesia.

Motion.—Intermittent muscular twitchings, subsultus tendinum provoked or aggravated by emotion. General loss of muscular power, giving rise to feebleness of gait. Affection of the sphincters is not present. Various disturbances of the general health accompany or result from the cerebral disorders.

The author believes that general shaking (commotio cerebri) is answerable for the early symptoms, and that the later ones arise from a meningo-encephalitis which results from material lesions, *e.g.*, contusions, ecchymoses, sanguineous effusions occupying solely or exclusively the surface of the cerebrum, and other parts of the encephalon. He therefore considers the prognosis a grave one in such cases, and cure little likely to result; in any particular case it is often impossible to give an exact prognosis. As in many cases in which cure follows after some weeks, or two or three months, the same symptoms are present as in those which do not recover, it is generally necessary to wait six to eight weeks, and watch the course of events before giving a prognosis. In cases where the cerebral troubles are profound, and accompanied by severe general symptoms, the prognosis is necessarily bad, and it is equally so in the cases where the symptoms come on late after a period of apparent health.

As regards simulation, the author lays stress upon the fact that in his experience the intellectual troubles and purely subjective symptoms are never grave without at the same time a corresponding gravity of the general bodily symptoms (objective) mentioned above. Exaggeration may be suspected, if there is no proportional correspondence between the two.

Rarely cerebral affections may develop secondarily to those of the cord.

With regard to injuries of the cord in railway accidents, the author has little new to add to the well-known works of Erichsen and Page.

The author pleads for the retention of the term "traumatic neurosis" on practical grounds, independently of any theory which may be attached to it. The expression conveys clearly the dependence of the symptoms on an injury. He distinguishes three forms: (1) the hysterical (2) the common form, called by French authors hystero-neurasthenic; (3) a form in which one special symptom predominates.

(1) Cases in which an injury has brought about the development either of hysteria pure and simple, or of nervous disorders for the most part of clearly hysterical nature. The author states, that although he has examined a large number of injured persons he has never met with a case resembling the type described by Charcot and Guinon; and only one case of hemianæsthesia.

(2) Variety in which paralyses, contractures, anæsthesias, convulsive attacks either do not exist at all or only in mild degree; the chief symptoms being those of a cerebral state which has often been described, *i.e.*, depression of spirits, restlessness, irascibility, incapacity for mental or physical exertion, loss of memory, asthenopia and insomnia, with attacks of nausea, faintness and palpitation, together with various disturbances of the heart and of the digestive organs.

(3) In which one particular symptom predominates, *e.g.*, cardiac symptoms or dyspnœa, or rarely delirium.

Etiology.—The traumatic neurosis is produced by an injury acting in conjunction with an emotion—terror. Traumatic hysteria may be excited by an insignificant wound, but although this is proved, cases of hysteria pure and simple are only exceptionally met with in the traumatic neurosis; cases of the latter have a more definite etiology in arising after an injury of special nature, which appears to play as important a part as emotion. In the author's experience of twelve years as a medical expert in such cases, he has never seen, except in one woman, the group of symptoms which make up the traumatic neurosis present in persons who have met with all sorts of accidents, wounds from knives and revolver bullets, partial strangulation and attempts at suicide in various ways. Out of a much smaller number (about

400) of cases of victims of railway and carriage accidents, explosions and falls, he has met with 100 cases of the traumatic neurosis. In many accidents of the former category the emotional disturbance must be almost as great as in the latter. The nature of the injury is, however, different, because in the second class of accidents there is a more or less violent shaking of the nervous centres. This commotion is certainly present in persons who have received a violent blow on the head, and these are precisely the cases which are affected in greatest proportion by the traumatic neurosis. The group of symptoms of the traumatic neurosis corresponds to the description that has long been given by surgeons of the nervous symptoms which follow head injuries. It may often be very difficult to decide whether the trouble is purely functional or is due to a gross lesion of the brain or its membranes. In those patients who have not sustained a blow on the head there is reason to believe that there has been a severe commotion of the brain, spinal cord or nervous system generally.

Emotion as a factor.—Most persons affected with the traumatic neurosis have been more or less terrified at the time of the accident; sometimes the terror has been so great and the wound so small as to make the former appear most important; but the author thinks that the influence of emotion has often been exaggerated. For those persons most terrified at the time of the accident are not those who suffer most frequently from the traumatic neurosis, while those who do so suffer have often displayed little emotion at the time. Perhaps the effects of emotion are aggravated when there is also more or less physical "commotion" of the brain. Again, the symptoms of the neurosis may appear in cases where from the sudden nature of the accident there can have been no time for emotional disturbance. The author does not consider that neuropathic tendency or predisposition plays an important part in the development of the traumatic neurosis; evidence of such a tendency is frequently absent, and the coincidence of injury and evident emotion is sufficient to produce the symptoms.

The statements in this paper appear to be fairly borne out by the details of numerous cases which are given.

Prof. Pel[66] reports the case of a carpenter, aged 27, with no family or personal history of disease; but with a large head from rickets or hydrocephalus. After a heavy fall (? on the head) which caused loss of consciousness, he complained of persistent localised pains in the back of the head on the left side, and subsequently of giddiness on movement, together with an overpowering tendency to sleep. The left half of the back part of the skull was tender to percussion. There was paresis of the right half of the body and left facial nerve. Power of vision was deficient. All these disorders were permanent and did not change, but in part were slowly progressive. Sensation was everywhere normal. The tendon reflexes in both lower extremities were exaggerated. Standing and walking was impossible, but when lying on his back in bed the patient had unimpaired movement of his legs. As the patient had a small polypus on the right vocal cord, which was caught between the cords on phonation, it was removed without any difficulty under cocaine. Immediately after the operation speech was lost, and attempts to speak only resulted in a wider separation of the vocal cords from each other; the levators of the epiglottis were also paretic.

Three weeks later there was noticed for the first time right hemianæsthesia, hemianalgesia, hemianosmia, hemiamblyopia with loss of taste on both sides. Two months after the operation the patient could only whisper feebly, and could scarcely put the mouth and lips into the positions necessary for forming letters. Subsequently he had two hystero-epileptic attacks.

Complete paralysis of the adductors of the vocal cords after such a harmless procedure as the removal of a small laryngeal polypus could only be explained as a "local traumatic neurosis," and strengthened the diagnosis already made of the condition previous to operation as that of traumatic neurosis. The appearance of right-sided anæsthesia and bilateral loss of taste without increase of the previously existing right-sided paresis, together with hysterical mutism and hystero-epileptic attacks, clearly indicated that the case was an example of the classical male hysteria or traumatic hysteria of French writers.

As to the further history of the case, none of the various modes of treatment tried, including hypnotism, produced any effect; the man died of a double catarrhal pneumonia which was fatal within a few days of the onset, and was a further proof of the small resistance offered by such a patient to the attacks of acute disease. Except for hydrocephalus the brain was healthy, presenting no abnormality which could account for the symptoms observed during life.

In conclusion, the author emphasises the fact that functional nervous disorders may follow not only on an injury, but the performance of an operation or any method of local treatment of an irritable organ, and this should be borne in mind, since the possibility of such an occurrence is apt to be forgotten in the present day, when attention is directed chiefly or only to the material consequences of surgical procedures.

Dr. Morton Prince[56] observes that great difference of opinion exists as to the prognosis in the traumatic neurosis, some holding that the tendency of all cases is towards recovery, and that at an early period; others that the pathological condition disappears if at all only after a prolonged duration. He therefore records two cases of twenty-nine and one of twenty-eight years' duration, which belong to the so-called hysterical type, are of the male sex, and have, so far as can be judged, maintained their original form without the development of secondary pathological consequences.

The first patient was struck by a piece of shell which did not injure the skin beyond a black and blue mark below the left shoulder blade. This left side was "paralysed immediately after the accident, and has remained so ever since." Dr. Prince saw him in 1886 and in 1891. At the first visit he suffered from anæsthesia of the left arm and leg only, at the second this extended over the whole left side of the body, except the face and palm. There was also left hemiparesis, and some deafness, loss of taste and of smell on the left side. Knee-jerks were both within normal limits. The patient stated that his condition varied from day to day. In the second case, one of gunshot wound, the ball entered the left arm between the upper end of the belly of the biceps and the deltoid tendon passing through the bone. There was, on examination in 1886, complete paralysis of all the muscles of the arm and hand from the shoulder down, including the deltoid. There was also slight loss of sensation over the limb; no atrophy or degenerative rigidity or contracture of the muscles, which were softer than those of the left arm from disuse. In the third case there was decided, but not absolute paralysis of the muscles of the left hand and arm, including the deltoid, without atrophy, rigidity, or exaggeration of the deep reflexes; there was also profound, but not absolute anæsthesia over the limb. The condition came on after recovery from twenty-four hours' loss of consciousness, consequent on injury from the wind of a round shot which passed between the extended arm and the body; it had remained the same until Dr. Prince examined him in 1886, and again in 1892 it was found unaltered.

The author concludes that the picture presented resembles that of functional disease, and that this diagnosis would have been made in cases presenting similar symptoms immediately after an injury; he sees no reason to question the diagnosis because of the long persistence of the symptoms.

Traumatic Neurasthenia.—Dr. Löwenfeld's experience in South Germany is that the working classes are affected by neurasthenia and not by hysteria, as the consequence of accidents. This traumatic neurasthenia is distinguished by the cause only, and not by the symptoms; but symptoms of cerebrasthenia predominate. There is no hereditary taint in these cases; often, but not invariably, an interval of freedom from symptoms intervenes between the accident and the onset of neurasthenia. Bouveret attaches more importance to nervous shock than to physical injury, but in many cases the latter is equally potent. The sufferers from traumatic neurasthenia never regain their full powers of work, but after a time are capable of doing lighter work.

The Traumatic Neurosis.—Löwenfeld remarks that Oppenheim and Strümpell are the chief advocates of the view that there is a distinct disorder of the nervous system arising from injuries—the "traumatic neurosis." Charcot held the extreme opposite view, that such cases were instances of hysteria—of "traumatic hysteria." Contrary to the statements of French authors, the existence of a specific "traumatic neurosis" was never generally believed in in Germany. The author was the first to indicate the existence of "traumatic neurasthenia," and of German authors, the first to show that a specific "traumatic neurosis" did not occur. Injuries may give rise, according to individual predisposition, to various nervous disorders. Chorea, epilepsy, paralysis agitans may follow an injury as well as hysteria and neurasthenia, yet in the great majority of cases, one of the two latter diseases is found or a combination of both—hystero-neurasthenia. These latter cases correspond to Oppenheim's "traumatic neurosis."

Other factors in the causation of such cases, besides the mental effect of the injury, must be considered. Charcot and Oppenheim consider the mental effect as the most important, and they are justified in so doing in a large number of cases, as is shown by the fact that the extent and intensity of the resulting nervous disturbance bears little or no relation to the severity of the injury. Yet wherever there has been a mechanical shaking (commotion) of the central nervous system, the influence of this on the head and spinal cord, must be of, at least, more importance than that of terror. Schmauss showed experimentally that commotions of the spinal cord which were without any gross change in the organ, resulted in destruction (degeneration) of nerve fibres. The neurosis under discussion does not appear at once, but is gradually developed from molecular disturbances. In slighter degrees the effect of injury shows itself as a functional change, an exhaustion of the nerve fibres, and this exhaustion may, in severe cases, lead to their degeneration. Sudden powerful shocks (commotion) in the central nervous system in man may produce similar effects, and it is therefore not correct to neglect the mechanical causes in traumatic nervous cases so much as Charcot and Oppenheim have done.

In severe injuries, without, however, actual damage to the nervous centres, there are the depressing influences of pain, with attendant loss of sleep, appetite, and fresh air, and anxiety with regard to future means of subsistence.

The fact that slight injuries, quite unimportant in themselves, may result in neuroses, can be explained only by taking into consideration the individual predisposition. The combined effect of bodily injury and mental shock may even in a perfectly sound man give rise to nervous disorders. In the cases in which moderate injuries to the body without mechanical effects on the central nervous system cause lasting neurotic disturbances, there must have been an antecedent neuropathic tendency. The more pronounced this neuropathic disposition, the more trivial may be the injury capable of giving rise to subsequent nervous disorders. This predisposition may be inherited or it may be acquired from alcoholism, masturbation, former accidents, injurious influences of mode of life or occupation. Lastly, the nature and seat of the injury may have an influence on the seat and character of the nervous troubles.

Dr. Knapp,[57] in discussing the question of "simulation in traumatic nervous diseases," thinks that the tendency has been to exaggerate the frequency of simulation in nervous affections of traumatic origin. The reproach of simulation is sometimes made because the examining physician is a general practitioner without any special training in or knowledge of nervous disease; to such a man a patient suffering from astasia-asbasia, a patient, for instance, who might be able to hop but unable to walk, would seem to present a clear case of simulation, yet an experienced neurologist would admit the genuineness of the condition. Again in hysteria, the nature of the conditions is often curious and improbable, *e.g.*, monocular diplopia; there may be extreme amenability to suggestion, so that the manipulations of a prolonged examination may accentuate or diminish the symptoms present; the influence of stimuli is often very marked. "When we prove that a patient, under a powerful stimulus, can do what she says she cannot, that her pain is most marked when her attention is called to it, that her condition varies from day to day . . . . . we do not disprove the existence of hysteria or of neurasthenia."

Certain symptoms are valuable; contraction of the visual field can with difficulty or not at all be successfully simulated. Anæsthesia, if the boundary is well defined, may be safely assumed genuine. If anæsthesia is simulated an *unexpected* painful stimulus will soon detect it. Differences in the skin reflexes in hemianæsthesia will establish the existence of anæsthesia beyond dispute. A *persistent* rapid pulse is of positive value, as is probably also an acceleration of the pulse produced by pressure on painful regions. The author thinks that the simulation of one of the more complex forms of traumatic nervous disorder cannot be successfully maintained through a long medical examination by a skilled neurologist. In fifty consecutive cases of all kinds which he has examined with reference to determining the degree of injury and the amount of compensation to be claimed, in only two was simulation probable, and one of these patients was a child.[58]

VIII.—General Features of Neurasthenia (Löwenfeld).

Stigmata of Neurasthenia.—Certain symptoms occur again and again with monotonous regularity in cases of neurasthenia. They are almost constantly present, and form the ground work of the disease or "stigmata," to use Charcot's expression. They are limited in number. Dizziness and pains in the head, inability for mental work, disturbed sleep and irritability of temper characterise the cerebral, pains in the back and weakness in the limbs, the spinal form of neurasthenia. Add to these palpitation, nervous dyspepsia and sexual weakness, and the list of typical symptoms around which others are grouped in varying number is complete.

Many symptoms were formerly allotted to hysteria, hypochondriasis and melancholia, that later observation has shown to be really due to neurasthenia, and in hypochondriasis a number of neurasthenic symptoms occur which are merely concomitant with the hypochondriacal symptoms and are really due to the accompanying neurasthenia. The author has found in his study of neurasthenia that the symptoms are not only subjective, but that there are a number of objective signs. The complaints and personal history of the patients afford an insight of especial value into their personality. He distinguishes clinically four types of sufferers.

Clinical Types.—(1) Those patients who preface their tale by saying that they will make a short statement of their complaints in order not to take up the physician's valuable time. This is the prelude to an account in fullest detail of their medical history from their earliest years. This is Charcot's *"l'homme aux petits papiers."*

(2) Those who are more reserved, wish the physician to find out for himself what is the matter with them, and have an innate suspicion that their bodily state and nervous symptoms are not in accord; they do not wish to prejudice the physician by unsolicited statements.

(3) Those who say that they enjoy absolutely good health except for one symptom, *e.g.* insomnia.

(4) Those who from anxiety or dejection often experience a certain constraint in speaking out to the doctor. Neurasthenia from excessive onanism, from a fall in social position, and traumatic neurasthenia.

Intellectual Disturbances.—Intellectual disorders are of three kinds:—

(1) In the least severe cerebral exhaustion leads to degradation of intellectual capacity through slight failure of memory, lack of power of attention, and ready fatigue after mental exertion.

(2) In more severe cases besides quantitative loss in the intellectual sphere, not only is long-continued mental work impossible, but the general usefulness of the sufferer is impaired.

(3) In the most severe cases the capacity for mental work sinks to *nil*, the sufferer leads a purely vegetative life. In practice these three grades pass imperceptibly one into the other.

The emotional disturbances are well known. Those in the sphere of will are briefly indecision, dislike of change, but also instability in opinion, neglect to maintain personal honour, or to fulfil a distinct promise, fear of physical discomfort, and the condition described by Mobius as "akinesia algera." The character undergoes deterioration, but Arndt's view of moral insanity in such cases is based on a misconception.

Uncontrollable thoughts (*Zwangsdenken*) divided into uncontrollable (*a*) ideas (in the narrower sense); (*b*) actions; (*c*) sensations; and (*d*) emotions.

(*a*) Questionings of *cui bono,* over the nature of things and the existence of God; fear of mental unsoundness, to be distinguished from the hypochondriacal form; syphilophobia, misophobia, "délire du toucher" (admirably described by George Borrow in the character of the literary recluse in "Lavengro"), fear of robbers, of thieves, of fire, &c.; fear that they may be impelled to commit suicide; echolalia, coprolalia. In many of these cases the mental symptoms predominate over the others, throwing them into the background.

(*b*) Uncontrollable sensations affect chiefly the head and sexual organs. They play an important part in the causation of many cases of agoraphobia.

(*c*) These, in the form of uncontrollable fears or anxiety, constitute a very important class. Such conditions of unfounded irresistible fear or dread are met with in a quarter of the cases. They completely overmaster the patient in the attacks, but are generally confined to one particular set of conditions, and they affect persons to whom in all other circumstances fear is unknown.

The best known is the often described agoraphobia; in the worst cases of this affection the patients are unable to leave their homes, but in many the companionship even of a little child, or the fact that another person is walking in front of them, enables them to proceed with confidence. There are other dreads too numerous to describe; we note as of special importance the idea of inability to perform some special action, and especially some public function, which may prevent priests, professors, advocates, or statesmen from carrying on their avocations. There is ever a morbid fear of becoming possessed by one of these dreads.

The author describes an "angor nocturnus" in which patients wake up in a state of terror, which lasts from five minutes to half an hour, is attended with shivering and sweating, and followed by great prostration. Feelings of undefined horror, of impending misfortune, "a horror of great darkness" frequently afflict neurasthenics. Various corporeal disturbances such as dimness of vision, pulsation of cephalic arteries, inability to swallow, dryness of the mouth, changes in the respiratory and cardiac rhythms, motor weakness, trembling of the legs or whole body, heavy sweats, retching, desire to urinate or defæcate, may accompany and often follow the psychical troubles, and entail a state of exhaustion which lasts for some time, and render comprehensible the determined avoidance by the patients of the conditions likely to induce an attack.

Such conditions arising in mentally sound persons from circumstances inadequate to produce them in the healthy may be traced: (1) often to an attack of real illness or of faintness, or qualmishness affecting the patient in church, in court, or in the street, and the dread of a recurrence leading to persistent agoraphobia or similar state; or (2) the first onset may have been a sudden dread of being overtaken by illness during some public function; (3) they may come on suddenly and inexplicably. The surroundings of the attack then become fixed in the memory, and when the patient is again in the same place act as a stimulus to provoke a fresh attack. An auto-suggestion of incapacity, a fear of breaking down thus occurs, and it is important to remember that it may be overcome by another (fresh) suggestion —for instance, if the patient can be made confident that the thing he considers out of his power can be accomplished and that he will not break down. Similar conditions of fearfulness, or want of confidence are probably experienced by nearly everyone at some time in their lives, but they persist and master the neurasthenic because he cannot recognise their groundlessness and throw them off as the healthy man does. In the consideration of these uncontrollable thoughts, actions, and conditions of dread are to be noted: (1) the marked disturbance of the bulbar, and especially of the vasomotor centres. The repeated alterations in the calibre of the vessels in vasomotor disturbances, thus frequently recurring, may lead by interference with cerebral nutrition to psychical disturbance. There is a marked state of abnormal reflex psychic irritability. (2) The resemblance of these neurasthenic brain conditions to the hypnotic state. (3) Loss of control or lack of will power in neurasthenic patients is important, as it prevents them from resisting the development of these "fixed" or uncontrollable ideas.

Some authors have regarded these peculiar mental states as abortive monomanias, or paranoia. The author agrees with Koch who thinks that they cannot be considered as mental disease because they leave the patient untouched in his general mental processes, perceptions and relations with the external world; they dominate but do not falsify his mental processes, and rather hinder than favour the development of a psychosis. The author has found that they are especially frequent and obstinate in cases of hereditary neurasthenia, but in no way confined to these.

Vertigo.—Attacks of vertigo with subjective sensations of sinking, staggering, falling, &c., are very common in neurasthenia;

the more severe attacks are often accompanied by nausea, obscuration of vision, noises in the ears, and pallor. Paroxysmal states of mental confusion or stupefaction with numbness, coldness and weakness in one or both extremities on one side of the body also occur. These attacks come on without definite cause, but some are connected with stomach disorders.

Subjective Painful Sensations.—Various sensations are felt in the head, of pain, emptiness, opening and shutting, pressure, &c. (cephalic sensations of Gowers). Pain most commonly occurs in the back or sides of the head, sometimes over the eyes, and often as a characteristic feeling of a tight band encircling the head (*casque neurasthénique* of Charcot). There may be hyperæsthesia of the scalp over the seat of pain. These cephalic sensations occur in 75 per cent. of cases, vary in nature and intensity in different cases and in the same case from time to time. The causes are inherited tendency to neurasthenia, attacks of meningitis in childhood, injuries to the head attended with shock or fright, onanism, alcoholism, and long-continued mental over-exertion. They are much more common than the often described rachialgia or spinal irritation. Spinal pains often alternate with pains in the head; when originating in sexual excess or abuse they are attended with mental states of peculiar anxiety. Sensory disturbance of vision apart from migraine is rare, but feelings of numbness, stiffness, or loss of power in the tongue are not uncommon, generally affect circumscribed parts of it, and may be accompanied by difficulty in speaking. Pains are common in other parts of the body, and may simulate the lightning pains of tabes dorsalis, but are rarely so intense, and are more fugitive in character, rapidly changing their situation. Further cases of neurasthenia are met with in which there are general neuralgic pains. The common feeling of weakness or weariness in neurasthenia is not due to motor weakness; it is felt most before the patient stirs. Weariness and muscular weakness are two symptoms of neurasthenia which often occur together, but in the author's opinion are not causally connected.

Hyperæsthesia.—Hyperæsthesia may be general or affect circumscribed areas of skin. The underlying organs, *e.g.*, breast, testicle, or muscles may be tender to pressure. In traumatic neurasthenia, hyperæsthesia and hyperalgesia are common, and may last after the other effects of the accident are gone. Sometimes there is increased power of localisation of sensations over the hyperæsthetic areas. In traumatic neuroses Mannkopf's symptom (increased rapidity of pulse on pressure over the tender areas) is sometimes present, but observations are still required to show whether this holds for ordinary cases of neurasthenia. On the other hand anæsthesia is rare, and then only affects small areas of skin; the anæsthesia found in cases of hystero-neurasthenia is to be attributed to the hysterical element in the case.

Attacks resembling Focal Sensory Epilepsy.—The author relates a case of a man, age 50, affected with neurasthenia for twenty years, and who for twelve years had suffered about once a year from attacks in which feelings of pricking or numbness began in the occipital region, and spread over the left side of the head and face to the left arm, left side of the body, penis and left leg; they lasted for three minutes, and were followed by a feeling of weakness in the same parts for a quarter to half an hour; examination detected some weakness of the affected parts. The absence of any sign of organic disease, and of any permanent nervous trouble during so long a period shows the functional nature of the attacks. He has met with attacks of similar sensations in other patients limited to one arm or leg, or to the arm and leg on the same side, and often attended with cephalic sensations. The resemblance is very striking to the similar attacks which occur in migraine. Migraine is not a direct symptom of neurasthenia, but the latter has a striking influence on the severity, frequency, and duration of the migrainous attacks, which are aggravated should a condition of neurasthenia supervene in a patient subject to them.

In one case the attacks above described, which resembled those of sensory Jacksonian epilepsy, ceased when the neurasthenic condition improved.

Eyes.—Neurasthenic asthenopia, or irritable eye. There is a difficulty of fixation, the object fixed soon becomes indistinct, and if fixation is persisted in, shooting, stabbing, or boring pains in the eye come on. Long standing cases of neurasthenia may suffer from pains during rest or on movement of the eyes, and occasionally from double vision. These visual troubles are not connected with errors of refraction.

The author finds that contraction of the visual field rarely occurs apart from asthenopia, and that it is, as a rule, absent in neurasthenia. Charcot disputed the existence of contraction of the field in neurasthenia. Oppenheim attached great importance to it in the diagnosis of the traumatic neurosis, but a number of other observers deny its occurrence in such cases. Wilbrand's[88] observations, however, which are the latest, correspond with Oppenheim's, and he points out that the divergent opinions may have arisen from different methods of observation. An objective symptom in the traumatic neuroses is that known as Förster's typus. The essential point in this contraction of the visual field is this, that if an object be brought from the periphery to the centre of the field, it is seen further away from the centre than when it is moved in the contrary direction. The pupils may show excessive mobility or alternating dilatation and contraction independent of light or accommodation. Inequality is generally transitory, but has been observed to last for eight or ten months in a patient who recovered (there was no sign of organic disease).

Motor Disorders.—Difficulty in balancing on one leg, or in standing still with the eyes closed and the feet together has been noted, and also a *pseudo-ataxic* gait described. The author has never seen a well-marked ataxic gait in neurasthenia, and considers that its description is due to errors of observation. The gait may be unsteady from feelings of vertigo, and neurasthenic patients often deviate to one or the other side in walking, but it is never distinctly ataxic. He thinks loss of power is often largely of sensory origin. Other motor symptoms are a tremor like that of Graves' disease; fibrillary twitchings of the muscles, especially in the eye muscles, or the tongue; involuntary short contractions of single muscles or groups of muscles, especially of the thumb muscles, which may be sufficient to move the arm or leg, and prevent sleep or cause a restless condition.

The superficial and deep reflexes are as a rule increased. The author emphatically states that the knee-jerk is never lost in neurasthenia; with one or two exceptions, this is the general opinion.

Speech.—Disturbance of speech commonly takes the form of paraphasia, inability to recall the appropriate word or phrase, tendency to change or put in a wrong word, *e.g.*, to say good morning instead of good evening. Often the interchanged word has no connection with, or even the opposite meaning to that intended. Spasm of the pharynx or a feeling of stiffness in the tongue may render speech difficult or indistinct. Unimportant words may be rapidly repeated several times without stammer, or there may be stammering. All speech troubles are worse after excitement or over-exertion.

Neurasthenia Cordis.—Neurasthenic affections of the heart are frequent, occurring in more than 50 per cent. of cases. The pulse rate in neurasthenia is, as a rule, from 80 to 100 per minute, but may be persistently over 100, with or without attacks of tachycardia, in which it reaches from 180 to 200. These attacks may come on after excitement, overwork, the use of coffee, or some error in diet, or without any assignable cause, and are in many cases ushered in by the presence of subjective feelings in the heart, and accompanied by faintness, feeling of impending death, excessive anxiety and necessity of emptying the bladder and bowels. The attack leaves behind it pronounced weakness of the heart, and a condition of general exhaustion, which only disappears after some hours. The author has not met with the severe forms of tachycardia in neurasthenia described by Bouveret, but does not deny the possibility of their occurrence. These attacks differ from true angina pectoris in the majority of cases in the lesser intensity of the symptoms. Slowness of the pulse (40 or 50 to minute), is only occasionally met with. Very seldom weakness of the heart, as a result of over-strain, anxiety, alcoholism, &c., is so pronounced as to give rise to fears for the patient's life. The patients sometimes suffer from "hyperæsthesia of the heart and vessels," in which they are conscious of the heart labouring, and the blood circulating down to the finger tips. Flushings, blushings, especially of the face, disturbances of vasomotor innervation, lead to temporary or lasting dilatation of vessels. Frequently changes in the temporal arteries, by which they become more prominent and tortuous, are seen in young men of from 20 to 30 years of age. This pre-senile change is to be regarded as a sign of neurasthenia.

Nervous Dyspepsia.—Two forms of nervous dyspepsia occur: (1) patients in whom the general nutrition is unaffected; (2) those who waste, lose colour, and show signs of general malnutrition. This difference is by no means solely connected with the duration of the illness. In the mild form there are the usual symptoms of flatulent dyspepsia, the patients do not completely lose appetite, but are afraid to eat for fear of exciting pain. Other troubles, such as interference with heart-beat and respiration may perhaps be referred to pressure from a dilated stomach, but are also in part due to reflex influences on the bulbar centres. In the severe form, the wasting and loss of strength in connection with digestive troubles may go so far as to cause fears for the patient's life. Complete loss of appetite and disgust for food is even more than pain a cause of insufficient nourishment. Further observations are needed to reconcile the conflicting statement as to deficiency or excess of HCl. secretion in the neurasthenic stomach. Mathieu attaches more importance to impaired motility of stomach than to anomalies of secretion, but the subjective pains and discomfort bear no relation to the insufficiency of motor power.

With regard to intestinal troubles (enteropathies), the chief are excessive production of gas and constipation or diarrhœa. The author enumerates the disturbances of general health, and of the bowels which follow from constipation, and is also inclined to attribute stomach dyspepsia largely to constipation, because of its preventing the proper emptying of the stomach, and of the relief which is often given by a free purge at the onset. Later in the course of neurasthenia the constipation increases, and spontaneous evacuation of the bowels is lost altogether; they become distended with gas, and later still attacks of diarrhœa from irritation alternate with constipation, and the large intestine passes into a chronic catarrhal condition. The author notices amongst the visceral neuralgias attacks of pains in the liver which resemble biliary colic. There is generally a striking increase of the knee-jerks; the attacks come on at remarkably regular intervals. In diagnosis, the fact that no inflammatory complications are found after the attacks have occurred for years, is important, and treatment directed to the nervous system is more efficacious than a course at Carlsbad.

Urine.—With regard to the urine the author notes the frequent occurrence of phosphaturia and of oxaluria, as dependent on disturbance of the general metabolism; he distinguishes two kinds of oxaluria: the one, in which the crystals are minute and do not give rise to local irritation, the other, in which they are larger and occasion pains or discomfort in perinæum and urethra, with frequent calls to micturition. He thinks no special diagnostic or ætiological importance attaches to the presence in the urine of the so-called urethral casts (*Urethralfaden or Tripperfaden*) which are made up of a ground substance of mucus with layers of epithelial or round cells in varying proportion, and often contain spermatozoa or oxalate crystals. These casts come from the urethra, or the prostate, and result from chronic urethritis.

Sometimes polyuria is met with, lasting for months or years and accompanied by excessive thirst, loss of appetite and symptoms of nervous dyspepsia. The author thinks thirst is the primary disturbance and polyuria the secondary.[1] In three cases of neurasthenia the author has found a trace of albumen in the urine, the amount varying slightly with the patient's general condition. Although it cannot be denied that a slight albuminuria may be dependent on neurasthenia, this conclusion should only be arrived at after every other cause for the albumen has been excluded. In badly nourished anæmic persons the albuminuria may be a consequence of the general condition.

---

[1] See paper above on "Hysterical Polyuria."

The idiosyncrasies so frequently met with in neurasthenia with regard to tea, coffee, tobacco, wine, food, and certain drugs are attributed to auto-suggestion.

Clinical Varieties of Neurasthenia.—According to the symptoms present neurasthenia has been divided into the following groups:—(1) cerebral neurasthenia or cerebrasthenia; (2) spinal, myelasthenia, spinal irritation; (3) cerebro-spinal or general; (4) cardiac neurasthenia (nervous heart weakness); (5) gastric (nervous dyspepsia); (6) sexual; (7) neuralgic form; (8) hemi-neurasthenia; (9) hystero-neurasthenia. These numerous divisions are to be explained by the fact that few patients complain of all symptoms, one especial group predominating; this is especially the case in nervous dyspepsia, in sexual and in cardiac neurasthenic conditions. In the latter condition, symptoms of general neurasthenia may be present, but those relating to the heart cause the patient so much anxiety that he forgets to complain of his other sufferings.

(1) By cerebral or spinal neurasthenia it is not intended to imply that the nervous exhaustion falls solely on the brain or spinal cord; the connection between the two is so close that, as both clinical and experimental observations show, neither can be affected alone. But in individuals the resistance to morbid influences varies in different centres, so that cases occur in which signs of cerebral or spinal disturbance predominate. In many cases of cerebrasthenia physical condition and muscular strength are unimpaired. In the so-called psychical form the symptoms vary; in a great number of neurasthenics psychical changes (in volition, sensibility, and idea) are present, and these psychical anomalies stand in very varying relation to the signs of general cerebral exhaustion, which latter may be in no proportional intensity to the increased emotional irritability, feeble volition, and hypochondriacal or melancholic conditions present. Marked changes in temper, disposition, and emotional excitability may occur with unimpaired power for mental work and for sleep. Symptoms of loss of control (*Zwangsaffekte*) may be absent in cerebrasthenia or be marked whilst the mental symptoms are slight.

(2) *Myelasthenia.*—Two symptoms predominate: (*a*) pains in the back; (*b*) weakness of the limbs, especially of the legs; they are not necessarily in proportionate relation to each other. Numerous paræsthesiæ are felt in the legs, spermatic cord,

bladder, and prostate. The author distinguishes the following clinical varieties.

(*a*) Spinal irritation; feelings of stiffness and pressure in the back with pains radiating to the seat, private parts, and limbs. These pains have a very depressing effect. (*b*) Neuralgic form; in which pains of lancinating character affect chiefly the extremities. (*c*) Sexual. (*d*) Pseudo-ataxic form. (*e*) Form affecting the cervical portion of the cord.

(3) In sexual neurasthenia the author lays special stress on the evil influence of masturbation, on account of its frequency, and also because it is often begun at an early age when the central nervous system is not fully developed. Those cases of neurasthenia are alone to be considered of sexual origin in which disturbances of the sexual organs are the only, or the predominating, symptoms. Cases of neurasthenia due to these injurious influences do not always take the form of sexual neurasthenia.

Cases of sexual origin are divided into:

(*a*) Those in whom the functional disturbances are wholly confined to sexual centres in lumbar cord. Damage to the sexual functions may reach the highest grade without affecting other parts of the central nervous system. In some patients spermatorrhœa and impotence do not interfere with bodily or mental faculties, whilst in others of neuropathic tendency general disorders are entailed.

(*b*) The symptoms of sexual disorder are associated with symptoms of neurasthenic affections of other parts of the nervous system, generally brain and cord.

(*c*) In which sexual disorders come on secondarily to general neurasthenia, *e.g.*, as result of a fever, and increase the nervous exhaustion from causing frequent seminal emissions. An irritable condition of the lumbar cord may, however, have been induced by inheritance or sexual abuses, and be first brought out by the action of a cause entailing general nervous exhaustion.

(4) *Hereditary Neurasthenia.*—According to Beard, a chief characteristic of hereditary neurasthenia is its obstinacy. Levillain divides these cases into three chief varieties:—(*a*) Neurasthenia complicated by hereditary symptoms (phobias, tics, *délire du toucher*); (*b*) Neurasthenia, brought about by accidental causes, which develops into hystero-neurasthenia; (*c*) Neurasthenia of distinct hereditary origin.

(*a*) The author says that in one group of cases it cannot be shown that the neurasthenic condition arises through an inherited neuropathic tendency. Other causes sufficient to produce it in those of healthy inheritance are present. In a second group the causes are complicated, and the influence of heredity is not always clear. In a third and very large group the influence of inheritance is manifest, neurasthenia developing under circumstances insufficient to produce it in the healthy. In a fourth group neurasthenia comes on without any definable reason. These individuals show signs of an abnormal condition of the central nervous system in childhood, such as headache, convulsions, attacks of migraine, night terrors, and somnambulism. They are often quick intellectually, but break down in an unaccountable manner on any extra strain. Any intercurrent malady leaves behind it an increase of the nervous condition. In the strict sense of the term hereditary neurasthenia should be confined to this last class.

There are no pathognomonic signs of hereditary neurasthenia, but uncontrollable ideas and emotions (Zwangvorstellungen and Zwangsaffekte), and the so-called phobias (agoraphobia, &c.) are especially frequent, attain a greater degree than in other cases, and persist after the other symptoms have been removed. Psychical anomalies are also most marked in this form. There is a "circular" or recurrent form in which the same symptoms observe a periodic recurrence, each period lasting for some days.

Cause and Prognosis of Neurasthenia.—Neurasthenia has no fixed course on account of the individual variations in the cause of the illness, in the circumstances and mode of life of the patient, and in the treatment adopted. One characteristic is the frequent occurrence of variations in the course of the disease, of remissions, of return without assignable reason of symptoms which have disappeared, much to the discouragement of the patient. When this occurs without assignable cause, it should be borne in mind that disturbances of menstruation in women and of the sexual functions in men are apt to be concealed from the physician. This variation in symptoms is especially frequent in hereditary neurasthenia.

Neurasthenia does not, with certain exceptions, shorten life, and it interferes with rather than altogether puts a stop to the duties of life. As a rule, the disease responds well to therapeutic measures, cure, or, at any rate, more or less relief being obtained. The patient's surroundings and mode of life are of most importance in the prognosis. If a man has to continue working day and night for his bread, or is exposed to constant anxiety in his business; if a wife or mother must go on nursing a husband or child in spite of exhausted strength, then the disorder will naturally be increased; if, on the other hand, the patient can be put under better conditions, recovery can be secured.

The duration of the illness is of more importance in prognosis than the degree of nervous exhaustion. Symptoms of short duration are easily cured, and *vice versâ*. As a rule, hereditary neurasthenia is especially obstinate, and a complete cure is altogether exceptional, but great improvement is possible. Of most influence in retarding recovery are insomnia, nervous dyspepsia, and nocturnal emissions.

There is no proof that organic disease of the nervous system can proceed from neurasthenic conditions, but the commencement of general paralysis often resembles neurasthenia, and here there are two possibilities, either that neurasthenia may lead to changes which pave the way for the further action of alcohol and syphilis on the brain, or that the neurasthenic condition is connected with the commencement of the changes of general paralysis. Not very rarely neurasthenia passes into a psychosis of functional character, most frequently into melancholia.

Myelasthenia does not give rise to tabes dorsalis; neurasthenic cardiac disturbances rarely, if ever, lead to actual heart disease, and the attacks of tachycardia are never fatal.

Neurasthenia may lead to alcoholism, morphinism, cocainism, &c.

On Diagnosis in Neurasthenia.—(1) The patient's statements must never be solely relied upon in diagnosis. A complete picture of the case must be formed out of the patient's antecedent history, the cause and development of his troubles, together with the results of a thorough physical examination.

(2) On the mental side of chief importance are failure of capacity in different fields of intellectual work and defective memory, especially for recent events. Special groups of incidents may be forgotten, and fictitious events unwittingly substituted for them. Fixed hallucinations do not occur; uncontrollable ideas (Zwangvorstellungen) do not interfere with general mental operations, nor falsify ordinary perceptions, but persist in the mind as a sort of foreign body.

(3) Pains in the head are seldom of great intensity or long duration. Percussion tenderness over the head is absent, except in traumatic cases.

(4) No persistent paralytic conditions in the limbs; this is especially true of the facial and oculomotor muscles; such paralysis is against the presence of neurasthenia.

(5) Persistent alterations in speech are absent.

(6) Inequality of pupils is found but with good reaction to light and accommodation.

(7) The knee-jerk is never lost; diminution of knee-jerks is also unusual.

(8) That an illness is of neurasthenic character will be the more certain the longer it has lasted without the development of symptoms of organic disease.

(9) From another point of view the changing character of neurasthenic symptoms is important, but this point must not be pressed too far for in certain organic diseases the symptoms may vary or disappear, and in neurasthenia some, such as the cephalic sensations, loss of mental capacity, sexual disorder, and nervous dyspepsia are very persistent. A sharp distinction of neurasthenia from hysteria is not practicable, because the symptoms of the former are so frequently associated in the same patient with those of the latter. There is, however, no difficulty in distinguishing the symptoms which belong to each neurosis if the so-called hysterical stigmata, *e.g.*, anæsthesias, hemianæsthesia, paralyses, contractures, different forms of convulsions, attacks of sleep, and hysterogenic zones are borne in mind and their presence sought for. No reliance is to be placed on the often stated inconstancy or fleeting character of hysterical symptoms.

IX.—Treatment of Hysteria and Neurasthenia (Lowenfeld).

The general aim of all treatment is of course to remove the cause, but in removing the local disorder which may have preceded for some time the general disturbance of the central nervous system one cannot always relieve the latter, and in many the former is an expression of the general nervous condition. If the changes in the nerve centres which form the basis of neurasthenia and hysteria have, whatever be the original cause, been kept up for some time, the removal of the cause does not insure the return of the centres to the normal state. That depends on the duration of the changes in question, on the constitutional powers of resistance of the nervous system, and on the presence or not of an inherited or acquired neuropathic constitution, and takes place if at all only very gradually. In many cases a perfect restoration of the central nervous system to its normal condition is no longer to be attained.

As regards diet the author is not in favour of the vegetarianism that has been recommended for these disorders, but thinks a mixed diet in easily assimilable form at not too long intervals is best. He restricts the use of coffee in cases of heart weakness. When neurasthenia has come on as the result of deficient nutrition, as the body-weight increases and nutrition improves the nervous symptoms dwindle, but in most cases no such result is to be anticipated, wasting being a secondary result of the nervous disorder. If persistent alteration in the composition of the gastric juice be present, in cases of increased secretion of acid the income of fats and carbo-hydrates must be restricted whilst the digestion of meat goes on well; bread containing dextrin for starch and the use of such fats as butter are, however, desirable. In deficient acidity digestion of proteid can be carried on in the duodenum, if not in the stomach, and meat need not be cut off, but merely restricted in quantity. The use of alcohol must be decided by the individual habits and the indications in each case. Patients who suffer from tendency to swooning and disagreeable sensations in the head with flushings of the face are better without it.

Occupation.—As a rule, the occupation must be interrupted in neurasthenia for a longer or shorter time, especially in the case of occupations which entail much anxiety or over-fatigue, or which fall heavily on one nervous function. The worst cases must give up their work permanently, but it is not generally wise to completely forbid the occupation altogether; it is better to restrict the amount of work so as to bring it within the patient's powers.

In hypochondriacal cases it is particularly important that they should have some occupation to keep their attention off their complaints. In pronounced myelasthenia stop physical work; in brain-exhaustion, on the other hand, it is within limits useful; but *excessive* muscular exertion is a hindrance to recovery in brain-exhaustion also.

Sexual intercourse must be regulated, as sexual excess is highly injurious. No general answer can be made as to the desirability of marriage.

Medicines.—Medicines can only palliate the symptoms and not cure them. The bromides, preferably the mixed bromides, take the first place, and are especially useful in conditions of nervous irritability, but are injurious in cases where weakness and exhaustion predominate. Protracted administration of bromides is inadvisable. The suitable dose is 20-30 grains, or 52 grains per diem. Bromo-camphor is useful for nervous palpitation: ·1 to ·2 grammes three times daily.

Quinine is useful, strychnine not; atrophine is good in spermatorrhœa or too frequent nocturnal emissions. Iron is often abused in treating states of nervous weakness, but where there is anæmia, or disturbance of nutrition is the primary and nervous disorder the secondary condition, it is useful. Valerian is of service in psychical excitement, tendency to anxiety, &c. Narcotics in order of usefulness are codeina, sulphonal and paraldehyde. A drug that deserves notice but must be used with care, and is not to be given in heart-weakness, is hyoscine; especially useful in hysterical convulsions, with almost instant effects, in small doses of $\frac{1}{10}$-$\frac{8}{10}$ milligramme. Of antipyrin, antifebrin and phenacetin, the first is the best in headache, especially in migraine, in neuralgic affections generally, and in "cephalic sensations." Phenacetin resembles the bromides in action.

Change of Air.—The beneficial influence of change of air and surroundings in these neuroses is well-known; in old protracted cases so far as change of air can do good it must act by bringing the patient under climatic conditions entirely different from his habitual ones, and this is especially to be attained in sea and mountain climates.

Sea.—Many cases do badly at the seaside suffering from increased irritability, loss of sleep and appetite, and general weakness. Sea air is especially indicated in cases of exhaustion from excessive brain work and similar causes in persons of good digestive powers, and in conditions of nervous weakness with insufficient blood formation. It is not suitable for severe cases of hysteria, and patients with marked psychic disturbance.

High Altitudes.—In a large group of cases mountain residence is useful, but not as the sole means of cure. Severe cases of old standing will not be improved by mountain, more than by any other air. However favourable the influence of the air may have been during the residence of the patients at a high altitude the old troubles will come back sooner or later after their return to their homes. Even permanent mountain residence may without other aids, bring about no change in the condition of the nervous system. For this group, mountain air is to be regarded as an after-cure, to consolidate and perfect the benefit received from other modes of treatment. The therapeutic effect bears no relation to the actual height. Improvement shows itself in decreased irritability, increased mental capacity and muscular power, in improved sleep, digestion, and sexual power, and in relief of palpitation.

Hydropathic Treatment.—The author attaches especial value to hydrotherapeutic measures. One of the most useful is the wet pack. On rising from bed the patient is enveloped, all except the head, in a wet sheet; the attendant rubs the body all over for one minute, and then rubbing in a dry, warm sheet follows. The effect of this treatment varies with the temperature of the water (50° to 81° F. is the most generally useful), and the roughness of the towel.

The general effect is a stimulating and invigorating one. Another method is the ordinary wet pack which may be followed by frictions in a cold bath for three to five minutes He also especially recommends a bath, taken as follows. The patient sits up to his middle in water, whilst an attendant dashes the water over his back and chest; he then rubs himself, or is rubbed all over with a loofah or rough towel, or in cases of paraplegic weakness the legs especially are rubbed. After this, the whole body is immersed in the bath; and, finally, a cold douche thrown over the neck and back. Duration of whole, six minutes. Temperature of bath 72° to 86° F.; of douche 8° to 10° F. lower.

Sitz baths are often useful, especially in habitual constipation, but contra-indicated in irritable conditions of sexual organs.

Sea-bathing.—Sea-bathing is contra-indicated in thin anæmic persons who cannot stand the loss of heat entailed by it, also in cases with either insomnia, much neuralgia, paræsthesiæ, palpitation, convulsive attacks, tendency to head congestion or in severe myelasthenic conditions. *Warm* sea-water baths have not received the attention they deserve, and often give good results in increased appetite and tissue change, and improved nutrition. They are partly sedative, partly stimulating.

Electrical Treatment.—Löwenfeld advises the application of a weak constant current to the head by very large electrodes for one or two minutes in cases of brain exhaustion, and in "cephalic sensations." As a guide to the management of the applications no unpleasant sensations should follow the sitting, if the manner and kind of application has been suitable to the case. He also attaches much importance to galvanization of the spine, not only in myelasthenia, but also in general neurasthenia. Currents to pass transversely from sternum to dorsal spine, or from navel to lumbar spine. The electrode on the spine is to be moved upwards and downwards, keeping it longer on any painful spot or over the part of the cord most affected. Electrodes to be 5 cm. by 10 cm., current strength 2-5 ma., duration five minutes.

In all cases of nervous disturbance of heart's action, abnormal cardiac sensations, præcordal pain and anxiety, angina pectoris and in nervous asthma, galvanization of the vagus, by placing the +ve pole on inner border of sterno-mastoid above the manubrium and the —ve below the lower jaw, with a current of ½-2 ma. for 1½-3 minutes is useful.

Galvanization of the lumbar part of the cord is advised for sexual disorders; and in some cases, in which there is no chronic urethritis an intra-urethral electrode is to be applied to the prostatic part of the urethra. The author endorses the use of general faradization as employed by Beard and Rockwell, and finds it especially useful in hysteria and neurasthenia with symptoms of profound exhaustion and in neurasthenia with defective nutrition, poor appetite and emotional depression.

Psychical or Moral Treatment.—(1) Negative, consisting in prohibition of mental over-exertion, removal of injurious influences, such as emotional excitations exerting an unfavourable effect, the provision of sufficient employment for mind and body adapted to the patient's strength, and in certain cases isolation.

(2) Positive. Here it is of prime importance that a thorough explanation of the nature of his complaint, namely, that it depends on functional disturbance and not on organic disease, should be given to the patient. He should be encouraged to look for recovery. These disorders are aggravated and maintained by the patient's fears for the future, fears of impending brain and cord disease. Dispel these fears and a material step towards recovery is gained. If he knows the nature of his disease the sufferer will not experience fresh alarm at every fresh symptom. At the same time he must be impressed with the necessity of self-mastery, of putting forth the whole power of will to overcome his pains and tendency to excessive anxiety, and must be made to understand that he is considered to be really ill. To laugh at these patients, to rally them on their healthy appearance, and tell them that their sufferings rest on imagination is only to court mistrust. Educated persons, now-a-days, readily grasp the distinction between "organic" and "functional" nervous disease; the uneducated may be helped by illustrations. Then the source of the nervous disorder must be indicated in order that the patient may avoid it in future. Patients should be placed amongst congenial surroundings. They gain ease and relief of mind if the whole mode of life, meals, drink, exercise, and occupation is regulated by the physician, who must not only give directions, but see that they are obeyed. Every detail of the treatment advised, however disagreeable, must be scrupulously carried out, and hydrotherapeutic measures are

a useful means of discipline to this end, and give the patient more endurance and greater confidence. The author endorses the value of the Weir-Mitchell treatment in the more severe forms of hysteria and neurasthenia, but thinks that the number of cases in which the full treatment is necessary is a limited one.

Prof. Erb,[69] in remarking on the undoubtedly great increase of the neuroses during the last half century, observes that if this increase is to go on at the same rate the outlook for the future welfare of the nations is a gloomy one. But there are, fortunately, still a great number of persons unaffected by these changes who must replace the worn-out brain-workers, and there is reason to think that increased knowledge of the danger will result in pure, healthy conditions of life, in new methods of education and care in training, by means of which the workers will be prevented from falling into the same state. The peasant, rather than the artisan class, are likely to supply the future brain-workers free from nervous conditions. Then, by careful and judicious training, the nervous system may be educated, developed, and adapted to meet without suffering injuriously the increased demands made upon it by the complex conditions of modern life.

With regard to the prevention of the neuroses judicious training of the nervous system and of the body as a whole is most important. It is chimerical to expect that the intermarriage of neurotic persons can ever be prevented. Infants and children must be better cared for, more judiciously trained, both mentally and bodily. At school, athletic exercises in the open air must be insisted upon, and sufficient time allowed for them. Undue pressure must be avoided, only necessary things taught, and non-essential requirements be given up. Care of the body and a thorough physical training is most essential, and a right proportion between work and leisure, of which latter a due share should be spent in the open air in physical exercises, is as necessary for men engaged in sedentary occupations entailing much brain-work as it is for children and youths at school. Girls must be educated primarily for being healthy wives and mothers. Restriction in the use of such poisons as tobacco and alcohol must be practised.

In a paper on the subject of treatment, Dr. P. Blocq[70] observes that when hysteria was considered to be of uterine origin, treatment was directed to this organ; afterwards it was treated as a general neurosis by anti-spasmodics; now the tendency is to mental and moral treatment, as the opinion gains ground that it is a psychical malady. Of the correctness of the view that hysteria is a peculiar modification of psychical action the author entertains no doubt. He tabulates the modes of treatment as :

Psychical by (*a*) isolation. The *rationale* of this treatment is the separation of the patient for a variable time from his customary surroundings. To be effectual it must be as complete as possible. The best conditions are obtained in a hospital or hydropathic establishment. Further experience has fully confirmed Charcot's teaching that there is no more effectual treatment for hysteria than isolation. Isolation acts partly passively in putting an end to the conditions which engender hysteria, actively in being exactly opposed to the psychical disturbance which characterises the neurosis. The fact of removing the patient puts a stop to the series of causes—for instance, the excessive solicitude and anxiety of the friends, their grief or terror at some of the manifestations of the neurosis, or the presence of other relations affected with nerve disease—which have aided the development and contributed to the persistence of the malady. Isolation hinders that want of mental concentration, that distraction which in M. Janet's view plays so important a part in the development of some of the symptoms ; whilst on the other hand it substitutes the idea of cure for the hysterical manifestation with which the patient's mind is occupied.

(*b*) *Hypnotism.* — Theoretically the tendency to increased "suggestibility" in hysteria, *i.e.*, for sensory impulses of any kind to evoke, by distubance of the normal processes of association, abnormal and persistent motor effects, would seem to contraindicate the employment of hypnotism because it also renders the patient more amenable to influence by suggestion, but practically it has a limited application for the relief of serious symptoms, in which other therapeutic measures fail. The inconvenience of increasing the patient's amenability to influence by suggestion is more than counterbalanced by relief of aphonia, paralysis and contractures by means of hypnosis. A certain number of hysterics cannot be hypnotised ; in others the symptom is relieved for a time, but soon returns, or is sometimes replaced by a more disagreeable one. In any case the rules laid down by Professor Pitres should be followed. (1) In all cases to obtain the patients' consent, and to wait until they themselves suggest a trial of hypnosis. (2) Never to hypnotise a woman except in the presence of others. (3) Only to give suggestions useful for cure.

Transference.—This method, in which by the application of a magnet unilateral anæsthesia, contracture or paralysis is transferred to the opposite side, is sometimes useful, for the symptoms when transferred are less tenacious, more amenable to treatment, and may even disappear completely after transference.

Metallo-therapy.—The application of metals acting by indirect suggestion is occasionally undoubtedly instrumental in curing various hysterical manifestations.

Hydro-therapeutics, of all external applications, are the most useful, and to be most often had resource to. Preferably cold douches 13-18° C., by a jet thrown first on the body, ending on the feet for 15-20 seconds once or twice a day followed or not by rubbing. The wet sheet may be employed where this method cannot be followed out.

Electricity.—Static electricity is most generally suitable.

Massage and gymnastic movements are both of great service. The above methods can of course be applied whilst isolation is being carried out.

Of drugs, the alkaline bromides are rarely of any use. The author, however, recommends in conditions of extreme agitation or mental excitement the bromide of camphor as follows:—Camphor mono-bromide 3 grammes, ext. of quassia 2 grammes, syrup of belladonna q. s.; divide into thirty pills; two to six per day. Valerian, valerianate of zinc, quinine may be useful, and if narcotics are necessary sulphonal and chloral.

Dr. Blocq divides hysteria into the following forms: (1) latent; (2) mild; (3) severe (grande hystérie, hystéro-épilepsie); (4) mono-symptomatic.

(1) Children, who, together with nervous inheritance, suffer from troubles such as somnambulism, night-terrors, attacks of violent temper, require to be most carefully trained, as they are likely to develop hysteria in later life. Physical exercise is to be insisted upon, intellectual work and emotional excitements interdicted. Teaching should not be begun until the age of eight or nine, and then conducted in the most regular and systematic fashion. If the parents are affected with nervous maladies, the children should be sent away to school.

(2) In the author's opinion isolation is rarely necessary, and hypnotic treatment absolutely contra-indicated in this form; whilst in the third variety they both find their most important applications.

(4) Cases of mono-symptomatic hysteria are especially troublesome on account of their tenacity. All the various forms of treatment are often tried in turn without success.

Treatment of Individual Symptoms.—In the convulsive attacks it is generally sufficient to watch the patients to prevent them from injuring themselves, but if they occur in series they should be cut short either by searching for and pressing upon the hysterogenic zones, or by the inhalation of ether or bromide of ethyl.

For brachial monoplegias, Charcot's method of causing the patient to make efforts with the dynamometer several times a day, and to note the smallest progress made is to be employed. In this way almost continuous progress may be made.

For contractures, massage, the application of metals, or of a magnet are often effectual. Surgical interference is only justifiable in the case of connective tissue adhesions. In the case of hysterical anorexia, if the prescribed meal is refused, food is at once to be administered by the nasal tube.

REFERENCES.

I.

1. "Ueber den psychische mechanismus hysterische Phänomene," by Dr. Joseph Breuer and Dr. Sigm. Freud (preliminary communication); *Neurol. Centralblatt*, Nos. 1 & 2, 1893 [See also Nos. 10 & 11, 1894.]
2. "Quelques définitions récentes de l'hystérie," par. Prof. Pierre Janet; *Archiv. Neurol.*, 1892.
3. Brachet. "Traité de l'hystérie," 1847, p. 204.
4. "L'Anesthésie hystérique, l'amnésie hystérique, la suggestion chez les hystériques," by Prof. P. Janet; *Archiv. de Neurologie*, Nos. 69 and 70, 1892.
5. "L'Etat mental dans l'hystérie," par le Dr. P. Blocq, *Gaz. des Hôp.*, Nov. 25, 1893.
6. "La Théorie psychologique de l'hystérie," par le Prof Grasset; *Nouveau Montpellier Méd.*, Nos. 44 and 45.
7. "Des Stigmates hystériques," by Dr. P. Blocq; *Gaz. des Hôpitaux*, Jan. 28, 1892.
8. "De la perception inconsciente," *Arch. de Neurologie*, No. 58.
9. "Thatsachliches und hypothetisches über das Wesen der Hysterie," von Dr. Oppenheim, *Berlin Med. Woch.*, xxvii., 25, 1890; Mobius, *Schmidt's Jahrbucher*, 1890, p. 141.
10. "De la nature de l'hystérie," by Dr. Cousot; *Bulletin de la Soc. de Méd. Mentale de Belgique*, September, 1892. *Neurolog Centralbl.*, January, 1893.

II.

11. "Akademische Rede zum Geburtsfeste des höchstseligen Grossherzogs Karl Friedrich, am Nov. 22, 1893." Von Dr. Wilhelm Erb, Heidelberg, 1893.

12. *Leçons du Mardi*, 1887, s 368

13. *Lancet*, January 9, 1892

14. "Ueber hysterisches Fieber," von Dr. H. Sarbó; *Arch. f. Psych.*, xxiii. 1891.

References in Dr. Sarbo's paper, 15 *to* 22.

15. "Etude sur la méningite tuberculeuse de l'adulte," Paris, 1884.

16. *Thése de Paris*, 1869.

17. *Thése de Paris*, 1883

18. "L'hystérie viscérale," Paris, 1883.

19. "Verhalten der Eigenwärme in Krankheiten," 1873.

20. "Ueber vasomotorisch. Innervationsstorung bei Hystérie"; *Allg. Wiener Med. Zeit.*, 1871, 23 and 24.

21. *Clin. Soc. Transactions*, 1886

22. Moravcsik. "Beitrage zur Temp., Puls., und Resp. verhaltnisse der hysterischen," Budapest, 1886.

23. "La polyurie hystérique," by Albert Mathieu; *Revue Neurologique*, October 15, 1893.

24. "De l'hystérie en Vendée," par le Dr. Terrien; *Archiv. de Neurol.*, 1893.

24*a*. *Brooklyn Med. Journ.*, April, 1893.

III.

25. "Paralysie hystérique systématique." (1) "Paralysie partielle ou systématique des functions motrices du membre inférieur gauche." (2) "Paralysie faciale hystérique," par J. Babinski; *Ext. du Bulletin de la Société Medicale des Hôpitaux*, Paris, 1892.

26. Ballet. *Bull. de la Méd. des Hôp.*, 1892.

27. "Astasia Abasia," by Dr. Philip Coombs Knapp; *Journ. Nerv. and Ment. Dis*, November, 1891.

28. Möbius. *Schmidt's Jahrbucher*, 1890, p. 25.

29. "Deux nouvelles observation de l'hystérie male," by Drs. Bourneville and P. Sollier; *Archiv. de Neurol*, 1891, p. 362.

30. "Un cas singulier de l'hystérie male," by Prof. Popoff, of Warsaw; *Archiv. de Neurol.*, 1891, p. 367.

31. "Sur trois cas de Monoplégie brachiale hystérique," by Dr. Kinnosuke Miena (of Japan); *Archiv. de Neurol.*, May, 1893.

32. "Ueber Akinesia Algera," von P. J. Mobius; *Deutsch. Zeitschr. f. Nervenheilk*, Bd. i., 1892, s. 121, quotes,

33. Neftel. *Virchow's Archiv.*, 1891, 3, s. 464.

34. "Zur Casuistik von Akinesia Algera," by Prof. Erb; *Deutsch. Zeitschr. f. Nervenheilk.*, 1892.

IV.

35. "Ueber die combination hysterischer und organisch bedingter Störungen in den functionen des Auges," von Prof. Arnold Pick (Prague).

36. "Die einseitigen centralen Sehstörungen und deren Beziehungen zur hystérie," von Prof. Knies in Freiburg.

37 Leber. *Handbuch der Augenheilk.* Bd. v., s. 985.

38. "Ueber die veränderungen des Gesichtsfeldes bei den traumatischen Neurosen," von Dr. H. Wilbrand; *Deutsch. Med. Woch.*, No. 17, 1892; *Neurol Centralbl.*, September 1, 1893.

39. "Zur Kenntiss der Augensymptome bei Neurosen," von L. v. Frank. Hochwart and H Topolanski Aus der klinik Prof. Nothnagel's "Beitrage zur Augenheilk." Heft xi.; *Neurol. Centralbl*, September 1, 1893

40. "Hysterical Anæsthesia with a study of the fields of vision," by Dr. John K. Mitchell and Dr. G. E. de Schweinitz (Philadelphia); *American J of Med. Sci.*

41. "A further study of Hysterical Patients and their fields of vision," by Dr. John K Mitchell and Dr. G. E. de Schweinitz (Philadelphia); *Journ. of Nervous and Mental Dis.*, January, 1894

V.

42. *Loc. cit.*

43. "Somnambulisme spontané dans ses rapports avec l'hystérie," by Dr. E. Mesnet; *Arch. de Neurol.*, 1892.

44. "Un cas de somnambulisme hystérique spontané et provoqué"; *Ann. Med. Psych.*, November, 1888.

45. Paper read before the American Neurol. Assoc, June, 1890. See also "Les Attaques de Sommeil Hystérique," by Gilles de la Tourette; *Archiv. de Neurol*, vol xv., No. 43, p 92; and "Narcolepsy; sudden periodical sleep seizures," Dr. A. W. Foote; *Dublin Med. Journ.*, 1887.

VI.

46. "Association de l'hystérie avec les maladies organiques du systéme nerveux, les nevroses et diverses autres affections," by Dr. J. Babinski; *Bull. et. Mém. de la Soc. Méd. de Paris*, November 11, 1892.

47. "Contribution au diagnostic différentiel entre l'hystérie et les maladies organiques du cerveau," Dr. Ghilarducci; *Archiv. de Neurol.*, 1892.

48. "Contractures organique et hystérique," par Dr. J. Babinski; *Ext. des Bulletins et Mémoires de la Soc. Méd. des Hôp. de Paris*, May 5, 1893.

49. "On the simulation of Hysteria by Organic Disease of the Nervous System," BRAIN, 1890, p 1.

50. See Buzzard. "Atrophy of the Optic Nerve as a symptom of chronic disease of the Central Nervous System," *Brit. Med. Journ.*, 1893, vol. ii., p. 779.

51. "Un case de sclérose en plaques et hystérie avec autopsie," by Prof. Grasset, Montpellier.

52. "Sclérose latérale amyotrophique ou amyotrophie hystérique? difficultés de diagnostic," by Prof. Charcot, *Archiv. de Neurol.*, March, April, 1893.

VII.

53. "Ueber die traumatischen Neurosen," by Dr. Adolph Strümpell (Erlangen); *Wien. Med. Woch.*, No. 23, 1893.

54. "La Névrose traumatique ; étude médico-légale sur les blessures produites par les accidents de chemin de fer et les traumatismes analogues."

55. "Ein merkwürdiger Fall einer traumatischen Hysterischen Neurose," von Prof. P. K. Pel, in Amsterdam; *Berlin Klin. Woch.*, June 12, 1893

56. "Three cases of Traumatic Hysterical Paralysis of 29, 28 and 29 years' duration respectively in males," by Dr. Morton Prince; *American Journ. Med. Sci.*, 1893, p 63.

57. "Simulation in Traumatic Nervous Diseases," by Dr. P. C. Knapp; *Boston Med. and Surg. Jour.*, vol. cxxix, No. 13, p. 319.

58. "The Traumatic Neuroses." Translated by Dr. Workman in the *Alienist* and *Neurologist*, from a paper by Dr. Seppili, of Imola, in "La Revista sperimentale ai Freniatria," 1891, January 11, contains a good account of the literature of the subject.

IX.

59. *Loc cit.*

60. Traitement de l'hystérie par le Dr. P. Blocq; *Gaz. des Hôpitaux*, May 21, 1892.

OTHER RECENT PAPERS ON HYSTERIA NOT ABSTRACTED IN THE FOREGOING:

"Syndrome hystérique simulateur de la sclérose en plaques," by Dr. Cochez; *Arch. de Neurol.*, 1892, p. 470.

"De l'œil hystérique," by Dr. Rouffinet; *Gaz des Hôp*, October 13, 1891.

"Les coxalgies hystériques," by Dr. H. F. Plicque; *Gaz des Hôp.*, June 6, 1891.

"La nutrition dans l'hystérie," by Drs Gilles de la Tourette et Cathelineau; *Le Prog. Méd.*, December 10, 1892.

"Deux cas de bégaiement hystérique chez les dégénérés," by Dr. L. Chaubert; *Le Prog. Méd*, Feb. 25, 1893.

"Hysterical Tremor and Hysterical Anorexia (anorexia nervosa) of a severe type," by Dr. Jas. Hendrie Lloyd; *American Journ. of Med. Sci.*, September, 1893. A review of recent French literature on hysterical anorexia and the account of a severe case of tremor and of anorexia neurosa.

"Ocular Psychalgia," by Dr. Smith Baker, of Utica, N.Y.

"Hysterical Mania," Dr. Van de Warker, Syracuse, N.Y.; *Amer. J. Med Sci.*

"Some points in the diagnosis of certain simulated Mental and Nervous Diseases," by Dr. J. T. Eskridge (Denver). Records cases of feigned epilepsy and mental disease by malingerers.

"Psychical import of variable Achromatopsia," by Dr. J. E. Purdon; *Transactions of the Medical Assoc. of the State of Alabama.* Observations on a well-known family of professional mediums who showed various derangements of accommodation in one or both eyes with curious defects of colour vision.

Reviews and Abstracts.

The Trophic Properties of Nerves.—By Justus Gaule, Professor of Physiology in the University of Zürich. An address delivered before the Physiological Section of the 63rd Congress of German Naturalists and Physicians. Translated by Mary W. Tanner.

THE trophic action of nerves plays a great part in the historical development of pathology. Numerous diseases have from time to time been attributed to disturbances of it; but these theories, often asserted and often denied, have never been generally accepted because they have never been experimentally proved. Neither with irritation nor division of nerves have physiological effects been obtained comparable with the pathological conditions which had been attributed to this trophic action. In presence of these negative results pathologists were forced to choose between two alternatives. Either they denied trophic functions to the nerves in their ordinary anatomical and physiological aspect, and associated it with particular kinds of nerve-fibres specially endowed with the trophic function; or they abandoned altogether the trophic function of nerves, and gave other explanations of the conditions which had till then been attributed to it.

In the course of the last two years I have carried out a series of experiments of which I hope to show the most striking, here and in the Physiological Institute at Erlangen; and the results are no longer so negative in character. In point of fact phenomena occur as a consequence of irritation of or injury to nerves (or, more properly speaking, to parts of the nervous system, for it is the ganglion cells rather than the nerve-fibres which are concerned) which must be looked upon as far-reaching causes of disintegration of the tissues, and which approximate, more than to anything else, to the so-called trophic disturbances of the pathologists.

Doubtless, in presence of these experiments, pathology will have to re-adjust the boundaries of its experience ; and I do not, therefore, think that the old views as to trophic nerves will be re-affirmed without further investigation. In the first place, it seems to me absurd to speak of special trophic nerves in the same sense as we speak of motor, sensory and secretory nerves.

I have found trophic action in both sensory and sympathetic nerves, and probably the motor participate in it. The trophic properties reside, then, in the whole nervous system, associated with the functions of its several parts, and, perhaps, connected with these, in various ways as yet unknown to us. This would suggest that the part which the sympathetic nerves play in trophic action is different from that of the sensory nerves, and this again is unlike that of the motor nerves. At all events the trophic action is not bound to any special kind of trophic nerve. It is only too evident that this latter hypothesis arose from the necessity of saving the trophic nerves at all risks. For when, in the case of nerves which could be anatomically demonstrated and physiologically examined, the result of the experiment was negative, then nerves were postulated, to which of course this experiment could have no application, seeing that they only had a hypothetical existence, and therefore could never be submitted to experiment. So pathologists will not, I hope, endeavour to explain their results in this way. But I should not wish to set aside other explanations of trophic disturbance which have, meanwhile, been attempted. Too much was hitherto attributed to the trophic properties of the nerves when it was believed that the pathological changes were caused only, and to a certain extent actually, by them. It would be absurd to suppose that external injurious influences may not act on an organism deprived of its trophic protectors ; and in so far as we have learned to take these influences into account, we have added something to our knowledge which we need never forget. But we must not overlook the fact that without this weakening of the organism's power of resistance, resulting from the failure of the trophic properties, these injurious influences could not have taken effect.

When a house falls we know that it is gravity which pulls it to the ground and destroys the whole building ; but we also ask what was amiss with the construction or the materials, for a well-built, let us say a "healthy," house resists the force of gravity. In presence, therefore, of the experiments which, after all, teach us to accept a trophic function in nerves, we must not rehabilitate the old views without consideration ; but we shall have to analyse anew the pathological phenomena, whilst not forgetting what we have learned meanwhile.

In order to arrive at a right comprehension of the trophic action of the nervous system, I think it well to make the following remarks. The preservation of an organism depends essentially on the fact of the forces developed in it and by it being always in equilibrium with the external forces acting on it through its surroundings. As soon as this is no longer the case the external forces will gain the upper hand, and destroy or at any rate modify the organism. But the evolution of the internal energy from the latent force (Spannkraft) available in the tissues and in the food is under the control of the nervous system, which so adjusts this evolution that it holds the external forces in equilibrium. These external forces fall into two categories, not so much with regard to difference of internal character, as to the impression they make on our sense organs. One class includes rapidly varying forces, the other those which are constant and equable in their action. The first are the best known to us, physiologically at least, because our sense organs are so constructed as to be especially receptive of rapid changes. These variations are met by corresponding variations in our own evolution of energy, *i.e.*, usually by movements; just as we physically resolve the variations in external forces into movement. This play of inner and outer forces is familiar to us because it goes on under the constant control of our sense organs. We have always credited the sensory and motor nerves with the task of protecting the organism in this struggle, *i.e.*, of inducing, at every variation in the dynamic condition of our surroundings, a compensatory setting free of energy in ourselves, which brings us into a new state of equilibrium. In the cases usually cited as examples this would be accomplished by purposive defensive movements. With that idea in our minds we generally forget that there is a second category of forces by which we are equally influenced, but of which we are unconscious because they are always acting. To these belong gravity, atmospheric pressure, temperature (which is only perceived when it varies, or when it exceeds certain limits), the surrounding vapour-tension, surface-tension, certain electric conditions of the atmosphere, &c. But these forces undoubtedly affect the organism, and as certainly must the development of energy in the latter be adjusted to them in order to resist them. This adjustment must be continuous, and we may be as unconscious of it as we are of the corresponding action of external forces; but if it is to result in the well-ordered economy of the organism, it must be under the control of the nervous system. This adjustment, then, I look upon as the trophic function of the nervous system. It is a constant, unperceived activity of the latter, about which we should know nothing if certain experiments did not show us what potent changes may result to the organism from any interference with it. If I wished to illustrate the relation which these trophic functions bear to the sensory and motor functions of the nervous system of which we are conscious, I might say that they represent the abscissa of the activity of our body, about which the movements set up by a stimulus and the other manifestations of energy fluctuate as more or less rapidly ascending and descending curves.

A connecting link between these two kinds of activities is formed, to a certain extent, by the protracted, slowly increasing influence of the nervous system on the muscles (for example, in the regulation of the capacity of the blood-vessels and of some intestines), and on the glands, &c., and we may regard these effects, according to their special character, sometimes as a new adjustment of equilibrium (as happens with a change of climate or of sea-level), sometimes as a slow variation.

Between these different functions of the nervous system there must exist internal relations, they must all be adjusted to each other; but further experiment alone can discover in what anatomical disposition, and in what physiological form these relations become evident.

At this point the following, at first sight weighty, objection might be made to these reflections. When we sever the nerve leading to an organ, say a muscle, we clearly separate that organ from the trophic adjustment, but it still remains under the influence of external forces. Now, if the previous considerations were correct, it ought to break down under this influence, and changes ought to occur in it similar to those brought about by experiments on the trophic nerve-centres. But that is not the case; the muscle does not alter, indeed in some cases it remains capable of contracting. This objection would hold good if it did not rest on a false premise, viz., that the condition of the muscle and its liberation of energy remain the same after the nerve is cut. But this is not so. I need only cite a few examples. A muscle so isolated no longer takes part in the growth of the organism, nor, if we exclude the growth, in the reparatory processes which preserve the organism from wearing out. If we injure the muscle it does not heal, and if we leave it to itself it gradually degenerates. A muscle severed from its nerve may retain its contractile power for a short time, but it soon loses it. It is evident that the supply of material which it possesses at the moment of division may yet be used for contracting, but it is not in a condition to make good what it has used up. Just that is wanting in the muscle whose nerve has been cut which we must look upon as the peculiar characteristic of a living organism, the power of self-repair ("das sich selbst Bilden"), and its capacity for liberating energy must therefore be quite different. A whole series of chemical changes is absent when we disconnect the muscle from its trophic centre, as we learn from the study of its products of decomposition; which show, besides a few qualitative differences, such great quantitative dissimilarity that, even when the muscle is richly supplied with oxygenated blood, the products form only a fraction of those of the living muscle.

We shall, therefore, not be wrong in concluding that with the division of the nerve the muscle is put into a different condition, in which, because of its qualitatively-different and quantitatively lesser metabolism, it is much less changeable and less susceptible to outer influences. No wonder, then, that a muscle still under the influence of its trophic centre shows, when the regulating function of the latter is disturbed by the experiment changes which on examination must be partly attributed to a rapid disintegration through external forces; whilst the same muscle is much less sensitive to external forces when, by dividing its nerve, it has been placed outside the influence of its trophic centre. This becomes still clearer when we consider a third condition of the muscle. A muscle with a severed nerve, is, it is true, not so quickly affected by external forces as a living one, still by degrees it is altered. It can dry up, soften, rot, &c., as muscle it will surely break down. When, on the contrary, we cut it quite out and place it in corrosive sublimate or bichromate of potash we can preserve its structure for years unchanged. What is the difference? Manifestly this. So long as the muscle is capable of metabolic action, and its substance can therefore be partly brought into a condition of decomposition, so long does it present to external forces (amongst which we must in this case reckon other life-germs), points of attack by means of which it can be destroyed; but if we completely kill the muscle, and, by hardening, bring its substance into a less changeable condition, then we render it unassailable by the outer influences to which it is usually susceptible. This should suffice to illustrate: (1) How many more points of attack the living muscle, with its constant self-repair, offers to outer forces; (2)

how great is the task of the trophic nerves of keeping it intact in opposition to these; (3) how extensive must be the changes which occur when (as at 1), the susceptible condition is maintained, whilst at the same time the regulation of the protecting force is disorganized. A complete cessation of the trophic action, which places the organ at once in the less sensitive condition, is clearly much less dangerous than an interference by which the active metabolism of the muscle is, it is true, retained, but the regulating power is disturbed.

The breaking of a valve in a moving locomotive can lead to the destruction of the engine and the whole train; but if the injury occurs while the engine is at rest, or if it stops it, then there need be no worse consequences than the breaking of a part of the machine.

Here I would draw attention to a point which is not directly brought out by the experiments in question, but which might present itself as an important factor when we have gained a deeper insight into these phenomena.

I have hitherto assumed that the trophic function consisted merely in preserving intact the whole organism and its organs in the struggle against the constantly acting forces of its surroundings; so that the breaking down of the materials present in it and the building up of new materials are accompanied by a liberation of energy which exactly balances the external forces. In point of fact the case is somewhat more complicated. An organism does not always remain the same, it gradually transforms itself. It has a period of growth, of maturity, of age, &c. During this process of transformation the relation of the separate parts is not identical. Certain organs increase in size in proportion to the others, some again decrease and disappear. We know that that occurs according to certain laws governing the whole metamorphosis of the organism. The trophic problem must therefore he extended, so that the liberation of energy in the separate organs shall be so carried on that the latter may not only maintain their status, but also remodel themselves in accordance with this general law. We must not misunderstand this in the sense that the remodelling itself is the work of the trophic functions. The causes of the transformation probably lie in the properties of the local cells; but for these to effect it without arriving at a condition of metabolism which would cause them to succumb to external influences, or would bring them into conflict with the whole vital economy of the organism, it must be under the regulating guidance of the trophic centres. This observation is necessary to explain why the latter, under certain circumstances, have additional work put upon them, and why the results of the experiment are enhanced. I have repeatedly found grounds for this, which I hope to work out later on. I will now proceed to speak of the experiments, which to a certain extent may be considered complete.

I began with Magendie's experiment, the intra-cranial division of the trigeminal. Magendie has described fairly correctly the changes which occur in the cornea after his experiment, and the long discussion which followed it would have triumphantly proved the trophic functions of the nerves if (1) the result had not been so uncertain; and (2) if Magendie himself, like most of his followers, had not fallen into the error of looking on the ensuing changes to a certain extent as actual operations working from within outwards; and (3) if the experiment had not stood too much alone. I have taken up the experiment again, and have endeavoured to make it conclusive. That is not such an easy matter, for as the knife works out of sight in the cranium, it is hardly possible, even with much practice, to know exactly where we divide the nerve, and the result depends on the position of the cut. But we can ascertain this by dissection after each experiment, and can compare the effects which occurred before the death of the animal, and then we learn that trophic changes occurred each time the cut was made in the ganglion, and did not occur when it was made outside it. Now, with the section outside the ganglion, the nerve fibres only were divided, while with that in the ganglion, not only were these divided, but the ganglion cells were injured; so we come to the important conclusion, which was a guide to me in my later experiments, that *it is the injury to the ganglion cells which interferes with the trophic action.* To this Eckhard has raised the objection that trophic changes occur when the trigeminal is divided above the ganglion, and he described a method of lighting on the nerve where it leaves the pons. I have followed his method, but on making a microscopical examination of this portion of the trigeminal, I found (in agreement with all the known facts of brain anatomy) that there are many ganglion cells in it. So in this case also it is these which are injured. If, on the contrary, we light upon the part which is seen under the microscope to be free from ganglion cells—that above the Gasserian ganglion, but not so near to the pons—then we get no trophic disturbance. This I considered as settling the point upon which the certainty of the result depends. As to the second point, there were weighty objections to the view, shared by Magendie himself and many others, that the changes in the cornea were to be looked upon in some measure as an inflammation actually originated by the trophic nerve. It is easy to show that external injuries are concerned in these changes, and that the inflammation may be almost entirely avoided by excluding them. Thus an opposite party arose who attributed the consequences of dividing the trigeminal to outward injuries alone. Because they found that the latter had some influence on the condition of things, they took them for the sole cause. At the same time, they quite forgot that an eye whose trigeminal—or rather whose Gasserian ganglion—has not been divided (for example, the other eye of the same animal, which has been exposed to just the same injuries), remains unaltered, while the eye on the side operated on is destroyed. Some change must have occurred in the latter as compared with the eye not operated on, by means of which external forces have got the better of it. There remained still another explanation. The eye of the side operated on is anæsthetic. Therefore, they said, because it was not aware of the outward injuries it could not protect itself by winking, &c. In this respect, the comparison between the eyes of which the trigeminal has been cut in the ganglion and those where the nerve was divided above the ganglion (therefore without injury to the ganglion cells), has been most instructive to me. Both eyes are anæsthetic, for sensation only depends on the nerve fibres which lead it to the brain. Neither eye, therefore, is capable of reflex action of the cornea, winking, &c. Both eyes can be injured by coarse mechanical accidents, such as a blow or the entrance of foreign bodies; and they are so injured. But when they are protected from this they act quite differently. The cornea of the merely anæsthetic eye remains almost as clear as before, but that of the eye which is also trophically injured soon becomes dim, deeper and deeper depressions appear in its epithelium, &c. The first is still quite a match for the steadily-acting forces, and is only altered by suddenly-varying actions, the latter is helpless against both. As the steadily-acting forces are always present, we need not wait, in the case of the trophically injured eye, for the occurrence of any special injury; the moment we harm the regulating ganglion cells the changes begin, and go on increasing with the summation of the causes. We can, therefore, give a different turn to the phenomena according as we remove from the cornea the action of one or other outer force. For example, we can prevent evaporation and cooling if we sew up the lid over the eye, and so abolish the effects of these forces; but as we cannot replace the actual regulation of energy by our protective measures the cornea will never remain normal. The alteration will always be evident on microscopical examination, and in most instances will be perceptible to the naked eye, and in each case it is in the direction in which the protection we supplied was either insufficient, or itself set other external forces in action.

When we compare these facts (1) in the normal cornea, which does not require artificial protection because the regulating energy of the animal suffices; (2) in the anæsthetic cornea, which only requires protection against rapidly-varying forces (movements, &c.); (3) in the anæsthetic and trophically injured cornea, which requires protection against these as well as against constantly acting forces; we see how the view of the trophic functions which I previously explained must have followed from my first experiments. This view has also been sustained by my further researches, though I cannot say it has as yet guided me to any positive knowledge; and there arise other questions into which I shall enter later on.

The next thing was to ascertain how far other ganglion cells possessed trophic functions, and on what part of the body they exercised them. I attacked the spinal ganglia because the Gasserian ganglion is looked upon as their homologue; and began with the frog, as an animal more amenable to experiment. It soon became clear to me that to arrive at any result the ganglion cells must be lighted upon somehow unawares, and I concluded from this that the instantaneous division of the Gasserian ganglion, which is inseparable from Magendie's method, was of the essence of the experiment. I could not at first explain the grounds of this otherwise than by the general reflection that the ganglion cells must be in a certain condition of life and activity to exert a trophic influence. In my latest experiments I noticed a fact which might help us to explain this circumstance. I observed in the sympathetic ganglia that a slight previous stimulus, for example, a lowering of temperature or a weak electric shock, would suffice to render them quite insensible to a subsequent experimental attack. Later on I discovered that this depends on a branch of the nerve of the ganglion, which when irritated entirely inhibits its trophic action, while after its division the ganglion is much more sensitive. Perhaps the insensibility of the exposed spinal ganglion depended on the circumstance that in the act of laying it bare, these inhibiting fibres were irritated. I emphasize the "perhaps," because fresh researches alone can inform us on this point.

With the spinal ganglion of the frog I got the best effect with small injuries made by a sharp knife in the second spinal ganglion (on the large nerve supplying the arm). The trophic changes occurred principally in the skin, and were of different degrees. Sometimes there was merely discoloration, at others there was a circumscribed bleaching of all pigment, so that white spots were formed. With more extensive changes depressions occurred in the skin, which arose from shrinking (combined with drying-up) of the epithelium; at other spots, on the contrary, there were warts, in which the layers of epithelium had increased, so that twenty and more rows of nuclei could be counted one above the other. In the most severe cases there were defects in the skin, which arose from a complete disintegration of the epidermis as well as of the cutis. I could not discover then, nor can I yet understand, why the mischief should be of such varying extent. Of course it might be that I injured the ganglion differently; but yet I had the impression that it depended greatly on the condition of the frog on which I operated. Freshly caught frogs, about twenty-four hours after capture, generally showed more serious alterations than those which had been kept longer in confinement. Neither could I make out why the changes (always with injury to the same ganglion) appeared now in one part of the skin, and now in another. Only this seemed certain, that their localization always coincided with the entrance of small nerves into the skin, and that the skin-lesions only occurred where (for example, on the breast) a cutaneous muscle was attached to the skin. Many fresh factors came here into play compared with the conditions in the corneal experiments; and we have added to our knowledge two well-ascertained facts: (1) that the trophic changes do not only occur in the sphere of the nerve whose ganglion is operated on, but over the whole body, and on the opposite side, so that the disturbance in the trophic functions must, anyhow, spread through the central organ of the nervous system to the origin of other nerves; and (2) that the division of nerve-fibres plays but a small part, for the most serious disturbances obtained when I did no more than remove a small layer of ganglion cells from the surface of the ganglion.

I was sorry to be obliged to leave the examination of the frog's spinal ganglia, for the time being, in this unfinished state. I say for the time being, for I propose to take it up again when I have gained more experience. But I clearly saw that I could not penetrate further into the question until I succeeded in getting sure and constant results to my experiments. I therefore turned my attention to the spinal ganglia of the rabbit, which offered much more constant life-conditions, from not being subject to the alternations between hibernation and spawning, land and water life, freedom and confinement. Dogs and cats, whilst in many respects desirable, were excluded from my experiments, because it is impossible to obtain the material in anything like sufficient quantity.

The experiments with rabbits were first of all arranged with a view to testing the best methods of procedure. It appeared that from the third, fourth, fifth and sixth ganglia disturbances arose in the skin, the glands and the muscles; and these, too, on the same as well as on the opposite side in the sphere of the ganglion operated on, and also in other nerve regions. Of these disturbances I selected those of the muscles, as being the most characteristic and the most easily produced. In these experiments on rabbits I also for the first time made use of stimuli: the making and breaking of a constant and not too weak current (about four to eight Daniel cells). I found an induction current ineffectual. These stimuli were about equal in their action to injuries with knife or scissors. Irritating with an acid had the same effect. The principle seemed to explain itself as follows. A stimulation of this strength certainly sets up a change in the ganglion cells; it is also an injury. On the other hand, the injurious action derives its character either from the parts destroyed or removed, or from the remaining parts. In the first case it is a negative action, in the second it is an excitation. Now, as a partial injury to the ganglion, where portions still remain, is a more powerful agent than a total destruction of it, it follows that the excitation must be the stronger. Subsequently I used either injury or stimulus as appeared desirable for a more exact determination of the circumstances. But one thing reappeared in all the experiments; injuries and stimuli were alike powerless when they acted on the exposed ganglion. The sac in which the ganglion is enclosed must not previously be opened. Perhaps this circumstance may in the future be explained by the presence of inhibitory fibres. Further, it was shown that the change which ensues in the ganglion cells exerts its action by spreading through the posterior root to the spinal cord; for if the posterior root is divided there is no action in the muscle. But we could not ascertain how the spreading into and beyond the spinal cord occurred; and for this reason, that we were not in a position to decide beforehand which muscles would be affected. There were many muscles in which the changes occurred, now here, now there, sometimes in numbers, sometimes singly. And so these experiments did not answer my expectations. It was only when I extended my researches to a sympathetic ganglion, the inferior cervical of the rabbit, that I was led to an experiment with such constant results that it could be used for a more searching study of the trophic functions. Here changes occurred, both with injuries and electrical stimuli, which always affected the same muscles, viz., the biceps and the psoas, which always appeared in the same part of these muscles, and were always of the same form and character. It could not, therefore, be doubted that a decided local relation existed between the ganglion and these muscles. To use a familiar expression, we may say that we have found in this ganglion the trophic centre of these muscles; for as we can functionally excite certain muscles through certain parts of the brain-cortex, so we can trophically modify certain muscles through this ganglion. I was strengthened in this opinion by an experiment, not often enough repeated it is true, in which, on irritating the first dorsal ganglion analogous trophic changes occurred in the triceps and semimembranosus. In these trophic centres the muscles must be represented in an arrangement differing greatly from their representation in the functional centres, and that is easily understood; and the fact that two such homologous muscles as the biceps on the upper, and the psoas on the lower extremity, should be governed by the same ganglion must suggest the idea that it has its origin in certain laws of embryonic development.

If the irritation of the ganglion only modified the muscle of the same side we might suppose that the paths along which the ganglionic influence passes communicate directly with the muscle as sympathetic nerves. But it as often happens that the muscles of both sides are affected, and just as often only the muscle of the opposite side, while that of the same side remains intact. This necessitates the presumption that the transference goes through the spinal cord. In point of fact a more exact examination shows that it is the branches of communication of the sympathetic with the spinal nerves, through which the action in a centripetal direction is led to the spinal ganglion, and thence on to the spinal cord. This circumstance explains how the earlier stimulations of the spinal ganglia, to which I alluded above, could originate similar, although more dispersed changes in the muscles. The effects issuing from the spinal ganglia were dispersed because they did not invade the centres themselves, but only affected the connections between these centres and the spinal ganglia, in which connections an exchange of paths may have already occurred.

Now what is it that goes on in the muscles when the sympathetic ganglia are stimulated? Till now I have been concise in the historical account of my experiments, because I was only speaking of things which I had described in greater detail in my contributions to the *Centralblatt fur Physiologie*, Bd. v., Heft 15, 16 and 22; Bd. vi., Heft 11, 13 and 25; Bd. vii., Heft 7. What I now have to communicate is for the most part new. Since my last communication to the *Centralblatt* I have succeeded in seeing the changes arise under my very eyes. I was indebted for this improvement mostly to the discovery of the inhibitory branch leading to the ganglion, to which I alluded above.[1] After becoming acquainted with this I was able by previous irritation to render the ganglion insensible, as well as by division of the nerve to make it very sensitive. As by this means I obtained better control over the stimulus, and could apply it when and how I liked, I ventured to expose the biceps on one or both sides by a slit in the skin, and whilst holding the limb in my hand I watched the result of stimulating the ganglion by connecting and disconnecting the battery (using non-polarizing electrodes). While before I could only verify the accomplished change by a dissection, I now saw it arise before me in all its phases. I must first remark that sometimes, but not always, the stimulation of the ganglion is accompanied by a convulsion of the animal, with contraction of the biceps. This appears to me to be partly due to subsidiary currents affecting the neighbouring cervical plexus. Whether the muscle contracts or not is immaterial to the further proceedings; it has, therefore, no relation to the actual changes. These first commence a few moments later, and we perceive that a spot on the surface of the muscle has lost its gloss and appears dull. A moment later fluid appears at this spot, and we begin to see that the surface of the muscle begins to sink under this spot, and becomes rough. This depression extends itself at the inner and outer sides straight across the muscle, while in the middle it reaches up into the fascia, from which it detaches a small narrow point. So, in from three, four, five, to ten minutes there arises a sore, always of the same characteristic form, the bottom of which quickly becomes red, while the walls thicken. Sometimes it stops at this point, but often the sore increases quickly in depth, so that it divides the muscle into two parts. In this case it is always accompanied by considerable hæmorrhage, which fills the wound, forces itself between the surrounding muscle fibres, and infiltrates the connective tissue. In my former communications, when I had only seen the completed change, I laid undue stress on this bleeding, and on the contraction of the muscular fibres. Now that I have watched the sequence of events these appear to me to bear a secondary character. The primary phenomenon is a softening or liquefaction of the muscular fibres at a definite spot (the localization of which perhaps depends on some detail of muscular growth). After softening, the muscular fibres tear, the result of the natural tension or tone to which they are subjected; and this without any contraction, or stretching, or spasm of the muscle. The leg can be held quite loosely in the hand and watched while this is going on. The fibres tear as a moderately stretched wire tears when it is made red-hot. It is a sudden local change in their elasticity, which deprives them of the firmness necessary to resist even the usual tension. When they are once torn the free ends must spring back, and thus arise the surrounding knots and the defined walls of the sore; the flow of blood into the latter follows as naturally as bleeding in an ordinary wound. The capillaries are too tender to bear stretching across the sore, so they tear too. Here we must take another fact into account: the ganglion is not without influence on the blood-vessels, and these are frequently dilated. This dilatation, although to a certain degree affecting the extent of the hæmorrhage, has nothing of a primary character, as determining what follows. It does not coincide with the muscular changes. The vessels may be dilated in several other places, in the whole skin, in many muscles, even in the same muscles, and, indeed, to such a degree that there may be slight extravasations; but the characteristic sores never occur except in the definite spots in the biceps and psoas. And on the other hand I have seen instances in which there was scarcely any dilatation of vessels, where the sores remained quite pale, and where, even under the microscope, no dilatation nor bleeding could be seen. If the sores are very deep then of course they cannot remain pale, they must bleed.

The microscopic examination shows in the earliest stages, therefore, when the sores just become visible, very peculiar changes in the muscular fibres; depressions form which fill with fluid, whilst the remaining substance lies, as shining, much-contorted bands, in the interior of the sarcolemma. After a time cell-formation and cell-transformation occur, which also, as I am assured by pathological anatomists, bear quite a special character; but which do not appear to me so important, as they also are secondary phenomena.

Let us appreciate clearly what has really been made out. The ganglion is irritated by an electric current which brings about a change in it. Almost immediately there occurs in definite spots, in definite muscles, a kind of dissolving of the tissue which leads to partial liquefaction, and which renders the fibres incapable of resisting the tension to which they are normally subjected. All the rest follows from this. The process is probably by no means so simple, but it agrees with the conception I sketched at the beginning; that the part which has been robbed of its normal power of liberating energy is injured by external forces. At the same time, it indicates that the ganglion cells exert an influence on this power of the muscle by means of which it maintains its metabolic activity in presence of all forces acting on it. It is not improbable that this influence rests upon an interference with the chemical economy of the muscle, for the sudden change in the muscle is more easily understood when regarded as a chemical action. Therefore, definite sympathetic ganglia take part in the chemical economy of definite muscular fibres co-ordinated with them. But how much more remains to be asked? How can we conceive this interference with the chemical economy? As a transference of material, or in some other way? What is occurring in the intermediate links? Does this influence go through the cells of the spinal ganglia and the motor cells of the anterior horns, and does it affect these or spring over them? After reaching the cerebro-spinal system by means of the branches of communication, will it be carried further in its centripetal and centrifugal fibres in the same manner and according to the same laws as are the functional (*i.e.*, the sensory and motor) excitations; and what relation does it bear to these?

These are all unanswered questions, which show that we stand only on the threshold of the problem. We can only advance slowly in so new a matter, because pre-conceived notions and the teaching of the schools leave us in the lurch; but, feeling our way in all directions, we must endeavour to furnish, by our individual researches, foundations on which we may further build up our knowledge. I have performed more than two hundred operations, so as to be able, with some prospect of certainty, to demonstrate the experiment, which I look upon, to a certain extent, as typical of trophic action. In the prosecution of these researches I have too often had to relegate the experiments to the category of the marvellous and the incomprehensible. Anyhow, these actions appear exceptional as long as our minds are prejudiced, but if we take the trouble to follow out the experimental origin of these theories of the schools, and to clear them from misconceptions, we find the contradiction vanish. I have felt compelled to work in this direction myself, and for this reason I have not hesitated to amplify the general remarks in my introduction.

Wertheim Salomonson on Tremor. (*Nederlandsch Tijdschrift voor Geneeskunde,* 1894, No. 1.)

THE author first gives a full account of the graphic methods of studying tremors and a description of the apparatus used in his researches, consisting of a system of two or three Marey's *tambours à répulsion*, which enables him to register every movement or tremor of a limb. He relates eight cases in which he obtained the singular tracings already mentioned in Gowers' "Diseases of the Nervous System," 1888, vol. ii., p. 595, Nos. 6 and 7. These tremors are called by our author allorhythmic or interferential tremors.

Lead poisoning, Parkinson's and Basedow's diseases, hysteria and alcoholism were the diseases of which this tremor was a symptom.

No relation could be discovered between the interferences and the respiration or heart's action; and no explanation of these strange tracings could be found in literature. The author was struck with the resemblance to the tracings (known in acoustics) of membranes swinging under the influence of tones of different pitch; and he supposed the alternating periods in the tracings to be caused by the movements of two different groups of muscles, affected with tremor of different amplitudes. By the following experiment he was enabled to verify his hypothesis. If a muscle is excited by a faradic current the interruptions of which are so slow as not to cause tetanic, but only clonic, contractions of the same duration as the interruptions of the primary current, it is evident that we can produce a regular tremor of this muscle.

By taking two faradic apparatus with slow interruptions of different rate and exciting two antagonistic muscular groups, we re-produce the condition of a member vibrating under the influence of two sets of muscles at different clonus rates. In this way the author experimented with both the sterno-mastoids, and thus obtained oscillations of the head, the tracings of which presented very distinctly and regularly the periods of interference.

That the amplitude of any tremor is determined by the mechanical conditions of the limb affected, can be easily proved by loading the latter with a small weight, whereby the amplitude of the tremor is increased.

Now the amplitudes of the tremors of different limbs in one patient are *almost* equal, and so the interferential periods are not so great as we could expect if the differences in amplitude were greater; and so we can understand that in pathology, if a member is vibrating under the influence of two different muscle groups, the difference in rhythm must be a result of the mechanical conditions of the member.

Another form of tremor, not yet observed or described until now, was seen by the author in a case of Parkinson's disease, and in a case of lead poisoning. He calls it "allorhythmic tremor in space."

In the first case in which the left hand shook continually, he constantly and regularly saw forty or fifty pauses per minute in the movements of flexion and extension of the wrist joint. During these pauses he could observe slight movements of abduction or adduction of the hand in the same joint. These movements were thus really alternating. The pauses were not always so marked as to obliterate every movement of flexion and extension. In this condition the movements were the result of flexion and extension combined with abduction and adduction, and a change in the plane of the movements could be discovered. Now and then these phenomena were less distinct when the forearm shook more strongly. In this tremor, also, could be observed an alternation between movements of flexion and extension and rotation, or movements of abduction and adduction.

The author also registered the tracings of these tremors, and here, too, got allorhythmic tracings in two planes. By combining these tracings and projecting them on a third plane perpendicular to the first two, he got tracings resembling those known in physics as the figures of Lissajous; which are produced by the combination of vibration in two planes, and which can easily be seen in using the kaleidophone of Wheatstone.

DR. VOORTHUIS (Sumatra).

BRAIN.

PART III., 1894.

Original Articles and Clinical Cases.

ON DISTURBANCES OF SENSATION WITH ESPECIAL REFERENCE TO THE PAIN OF VISCERAL DISEASE.[1]

BY HENRY HEAD, M.A., M.D.

PART II.—HEAD AND NECK.

INTRODUCTORY.

IN a previous paper[2] I pointed out that visceral disturbances produced pain at certain points on the surface of the body, and that this pain was frequently accompanied by more or less definite areas of superficial tenderness. But in this first communication I neglected all pain and tenderness above the level of what I called the area of the first dorsal segment. In the present paper I intend to treat of similar phenomena above this level, and to describe the situation of pain and tenderness in the head and neck produced by visceral disturbances.

An enormous literature has grown up around the subject of headache, reaching from the earliest times up to the present day. In almost every case the observer has noted the position to which the headache was referred and has subsequently classified the observations so obtained according to the disease from which the patient might be suffering. If I shall appear to neglect this mass of observations my excuse must be that they mostly relate to the position of the pain.

---

[1] Read before the Neurological Society, July 12, 1894.

[2] BRAIN, 1893.

Now the locality of the pain is instructive only when we know the area of tenderness with which it is usually associated. This tenderness, which is necessary to the full understanding of the locality of the pain, has been neglected, or at any rate dealt with in such a fragmentary manner as to be useless. Moreover, in order to estimate the value of any one factor in the causation of headache, the examination must be of a completeness impossible in ordinary practice. For if the eye surgeon notes the position of the headache in those patients who come to him with errors of refraction, he will come to the conclusion that almost any part of the head may be affected. In the same way the general physician will find that affections of the stomach will be accompanied by headache in equally numerous situations. Now in each instance the attention of the observer is concentrated on one organ only, and he notes the one positive detail but does not exclude the possibility of other coexistent, but independent causes.

The system I have adopted in determining the areas associated with any particular organ of the head has been to first exclude all pain and tenderness in the thorax and abdomen; then I noted the presence or absence of general states, *e.g.*, anæmia; lastly I excluded disturbances of other organs than the one under observation. After rejecting the cases which did not satisfy these tests a certain number of observations remained pointing directly to a relation between the organ in question and certain areas of tenderness. After determining that certain pain and tenderness was due to disturbance of a certain organ, the results of treating that disturbance were noted, *e.g.*, the effect on the headache produced by correcting an error of refraction. If the correction was successful it formed another link connecting a certain area of tenderness with the disturbance in a certain organ. Thus it is not sufficient to say that a headache is due "to the eye" or "to the teeth" unless we note the direct effect produced by treating the eye or removing the teeth.

On the other hand, when determining the relation of pain and tenderness in the head and neck to disturbances of thoracic and abdominal organs the converse precautions were taken. It is not sufficient to note that a certain headache appeared in a patient suffering from some thoracic or abdominal lesion, unless his eyes, ears, nose or throat had been examined to exclude the possibility that the headache might be due to disturbance of some one of these organs.

Thus throughout this laborious investigation completeness of record, rather than number of cases, has been the aim. Moreover an enormous number of observations made at the beginning of each new phase of the investigation were subsequently rejected, owing to an incompleteness in the records which must necessarily mark the stage of advance. Thus it is useless to say that the work is based on so many hundred or thousand cases, for if any advance has been made the earlier records must of necessity be incomplete, and if no advance has been made, why multiply cases?

[illegible] is paper I attempt to show the following points :—

[illegible] ch organ in the head stands in relation with one [illegible] as on the surface. To these areas pain is [illegible] ver them the skin may become tender, when [illegible] lition of that organ is disturbed.

[illegible] s are the nose, the eye, the ear, the teeth, the [illegible] alivary and other glands, the tonsils, the larynx, [illegible] stly, the brain itself. (Chap. ii.)

[illegible]) I then treat of herpetic eruptions on the head and [illegible] and attempt to show that in certain cases they corre-[illegible] d fairly with the areas of tenderness produced by visceral [illegible] ase. But, owing to the tendency of such eruptions in [illegible] head to involve several areas, the distribution of herpes [illegible] the head and neck is not of the same corroborative value as that of herpes on the trunk and abdomen. (Chap. i., § 2.)

(3) These areas do not for the most part overlap to an extent that can be appreciated clinically. Some of them, however, overlap distinctly, especially such as lie in parts of the neck where compression has taken place. But however great may be the overlap, it never even approximates to that which Sherrington[1] showed occurred in the distribution of every posterior root. (Chap. i., § 4.)

---

[1] *Phil. Trans.*, vol. clxxxiv.

(4) I attempt to map out the distribution of the fifth nerve by observing the results produced by destroying its branches in man. An examination of the results shows that my tender areas do not correspond to the distribution of the branches of the fifth nerve. (Chap. v.)

(5) As elsewhere in the body, the serous cavities and superficial structures do not cause referred but local pain. Thus an affection of the dura mater causes as local a pain as inflammation of the pleura or peritoneum, and injuries to the conjunctiva or anterior layer of the cornea cause no more referred pain than affections of the skin. (Chap. iv., § 1.)

(6) The phenomena of true megrim are then touched upon, and I attempt to show that it bears no relation to the phenomena of referred pain. Not only is it unaccompanied by superficial tenderness, but the pain follows l[illegible] differ from those of referred pain. (Chap. iv., § 2[illegible]

(7) I attempt to show that affections of the [illegible] abdominal viscera not only produce pain an[illegible] the body, but also cause pain and tenderne[illegible] areas of the scalp. The position of the he[illegible] governed by the dyscrasia or, directly, by the [illegible] abdominal organ which is affected. It stands, howe[illegible] direct relation to the position of the area of tenderness [illegible] surface of the body. Thus we must not speak of "anæ[illegible] or "cardiac" headaches, but rather of the headache c[illegible] lated to this or that dorsal segmental area. Whateve[illegible] the organ affected the headache and scalp tenderness wil[illegible] in the same position, provided the tenderness on the bo[illegible] occupies the same areas. (Chap. iii.)

(8) Finally, I treat of certain theoretical aspects of this work, and attempt to localise the central connection of these areas with the nervous system. At the same time an attempt is made to split up that complex of nerves known as the sensory part of the fifth into its elements, and to assign the great vagus nerve to its place in the cranial nerve group. (Chap. vi.)

To facilitate reference, the following indications may be found useful by those interested in special departments, touched on in this paper:—

Ophthalmology.—Chap. i., § 1 (p. 343 to p. 353); chap. ii., § 1 (p. 394).

Dental Surgery.—Chap. i., § 1; chap. ii., § 2 (p. 406).

General Medicine.—Chap. i., § 1; chap. iii. (p. 436).

Anatomy.—Chap. i., § 1, 3 (p. 386), and 4 (p. 391), chap. v. (p. 469), and vi. (p. 475).

CHAPTER I.

TOPOGRAPHICAL.

§ 1. *The position of the Areas of Tenderness in the Head and Neck.*

A. *The Dorsal Areas.*

When an organ in the head is diseased, the pain produced is frequently associated with superficial tenderness. Organs in the head behave in the same way as organs in the thorax and abdomen, and produce not only referred pain but also tenderness of the superficial structures in direct association with the referred pain. In my previous paper the methods of marking out these tender areas were described, and the characteristics of the areas discussed. Excepting in the region of the limbs these zones on the body fell into very simple forms. In the head and neck, on the other hand, a profound disturbance has been produced, and the primitive arrangement has been fundamentally altered by the shifting of the elements of the head on one another. I shall therefore begin by describing those areas which lie above a certain line. This line starts in the region of the nose, follows the lower margin of the bony orbit, runs directly back from the orbital process of the frontal bone to the upper part of the insertion of the ear, and then dips to form a curved line ending below the occipital protuberance.

Several methods are useful for testing superficial tenderness on the scalp, face and neck. On the forehead and hairy scalp, where there is a firm bony substratum, I use the blunt end of a pin; this is felt as a blunt touch until the tender area is reached. The patient then complains either that the touch hurts and the skin is "tender," "like a bruise," or else he may say that he is being pricked.[1]

If the tender area lies over the hairy scalp, a useful method is gently to pull upon or lift the hair. This is perfectly painless in the normal human being, but becomes painful if superficial tenderness is present over the scalp. Thus patients suffering from referred pain and tenderness will complain that their hair is sore when they brush it in the morning.[2]

On the cheeks there is danger that the head of the pin may exert pressure upon tender underlying structures, such as the teeth, and it is therefore advisable to support the cheek with the finger placed inside the mouth.

In every case it is best to use at least two methods, and to state which form of stimulation has produced the results figured on the recording chart.

The method I adopt in working out a case is as follows: —First the patient is asked to point out the situation of the pain. Then I rapidly test the skin, beginning from a part of the head over which sensation is unaltered, and working towards the hyperalgesic patch. Marks are made to show the points at which the tenderness begins. Then the history is taken or the patient engaged in conversation. After a time a second estimation is made. If the case be one of disease of the eye, ear, &c., I make a third estimation after the patient has been subjected to refraction testing, ophthalmoscopic or laryngoscopic examination, &c. It is very important neither to tire the patient, nor on the other hand, to concentrate his attention on the patch of tenderness. Over the head and neck marks made on the skin are fortunately invisible to the patient himself. The limits of the area should not be measured until the whole examination is finished. The areas are then transferred to a chart, or in the case of a variable part like the neck to a drawing made on the spot. The edges are either very roughly shaded, or else marked with greater definiteness by lines, according to the degree of accuracy with which the patch of tenderness could be defined.

---

[1] A word of warning is necessary with regard to the shape of the head of the pin. The pins I use have spherical heads, whilst the heads of the usual pins of commerce are segments of a sphere only. This difference is of no importance when testing tenderness on the body, for the pin can always be applied at right angles to the surface. On the other hand it is not so easy always to apply the pin at right angles to the surface of the scalp or forehead, and thus if the ordinary pin is used stimulation may inadvertently be made with the edge of the head, which is definitely sharp to the normal skin. To avoid this source of error I choose a pin with a spherical head which can never seem anything but blunt to the normal skin however obliquely the pin may be held.

[2] Women who have changed the method of dressing their hair suffer from tenderness of the scalp for some time after the change. They do not, however, react to the blunt head of a pin.

The bald scalp would at first sight appear an excellent field for tracing the limits of these areas. However, with the fall of the hair, the scalp seems to become less sensitive. Thus, though the shaved scalp forms a superb field for investigation the naturally bald scalp is comparatively unsatisfactory.

On those parts of the head and neck which are free from hair, heat and cold will also serve to differentiate the areas of tenderness ; but this method is clumsy, and the limits cannot be so clearly defined as by the other methods. It is however, theoretically interesting as showing the existence of exaggerated sensibility to heat and cold over these tender areas.

There was comparatively little difficulty in determining the relation of the areas on the trunk to the central nervous system, and I therefore felt justified in replacing the cumbrous regional names by names implying a relation to segments of the cord. With the areas of the head and neck this is impossible, except on purely hypothetical grounds, and I shall therefore give each area a name which in no way implies connection with any part of the nervous system, but simply relates to its regional distribution. In a subsequent chapter I shall attempt to show how far we are justified in assuming that these areas bear any relation to the segmentation of the upper part of the nervous system.

Fronto-nasal.

This area forms a racquet-shaped patch with the larger part lying over the forehead. It extends for from 2 to 2½ inches (5 to 6·5 cm.) above the root of the nose, reaching the junction of the hairy scalp with the forehead. It meets its fellow of the opposite side excepting just over the root of the nose. At the level of the eyebrow it extends out for from ¾ to 1 inch (2 to 2·5 cm.) from the middle line of the forehead. A downward flap occupies the side of the nose, but does not reach the middle line and ends below, above the upper part of the ala nasi (fig. 1).

The maximum point of this area lies over the orbital ridge of the frontal bone about ½ inch (1·5 cm.) from the middle line of the forehead.

The pain is said to be an aching "over the middle of the forehead," and the patient points to the left or right of the middle line, over the supra-orbital notch.

This area becomes tender in affections of the substance of the cornea, anterior chamber of the eye, upper part of the nose and incisor teeth of the upper jaw.

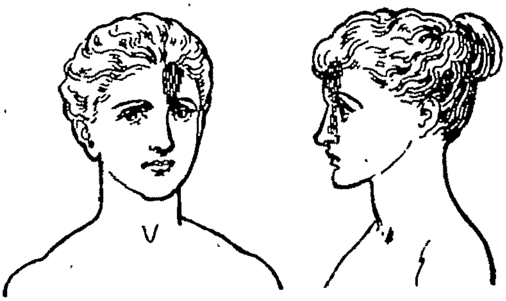


FIG. 1.

To show the area of superficial tenderness in a case of so-called serous iritis (Case No. 1). The tenderness follows the fronto-nasal area.

Case 1.—Harriet Q., aged 19. (Mr. Tweedy.)

She has had weak eyes since she was young, but they have been much worse during the last two months. She had attended the eye department on many occasions with similar attacks to the present one. The present attack began two weeks before I saw her.

The left anterior chamber is deep. Keratitis punctata and serous iritis. Slight ciliary injection. Tension of the eye as a whole normal. Right eye quiet but shows signs of past interstitial keratitis.

She complains of pain over the left side of the front of the forehead. Definite superficial tenderness as in fig. 1.

No pain or tenderness elsewhere in the body or head. No affection of the nose.

Menstruation regular and painless. Not anæmic.

Appetite good. No pain after food. No vomiting.

The next day the tenderness was equally well marked, but it gradually faded and had disappeared at the end of a week.

Mid-orbital.

This area, when fully developed, consists of two portions separated from one another. The lower part is roughly triangular, about 1 to 1½ inches (2·5 to 4 cm.) in height, by about 1 inch (2·5 cm.) across the base. It is situated over the centre of the eyebrow and involves the greater part of the upper lid. It sends a downward flap which occupies the inner (nasal) half of the lower lid. The second portion of this area is situated over the extreme front of the hairy scalp about 2½ inches (6·5 cm.) above the level of the root of the nose, and exactly above the patch on the forehead. The posterior limit of the scalp portion is about 2 to 3 inches (5 to 7·5 cm.) in front of a line drawn vertically from ear to ear. It is exactly over the fringe in a woman. The two portions are mostly distinctly separated from one another, but when this area is very well developed the upper part of the patch over the eyebrow comes very close to the anterior portion of the area on the front of the scalp (fig. 2).

A word of warning is necessary here. Directly we begin to work within the limits of the hairy scalp the boundaries of the tender zones cannot be determined with the distinctness that is possible over the forehead. For, owing to the presence of the hair, it is difficult to mark out the limits of the tender patch, and to see how nearly the second estimation resembles the first.

The more important maximum of this area lies over the upper part of the upper lid, just below the centre of the eyebrow. A second maximum exists over the patch on the front of the hairy scalp. In the large majority of cases the maximum over the centre of the eyebrow only, is present.

When this area is affected the pain is said to be "at the back of the eye" or "over the middle of the eyebrow."

The eyes are said to ache, and sometimes the patient complains of soreness "over the front of the hair."

If asked to point to the position of the headache when this area is represented on both sides of the forehead, the patient usually places the right hand over the front of the forehead. The thenar eminence is over the centre of the right eyebrow, and the fingers touch the middle of the left eyebrow. Thus one might easily be misled into thinking that the headache was truly "frontal." But when the patient is asked to point with two hands to the position of his headache, he places the tips of the fingers of each hand over the middle of the eyebrow on the respective side thus showing that the headache is not really in the centre of the forehead. This source of fallacy has not been recognised by those who have studied the situation of pain only.

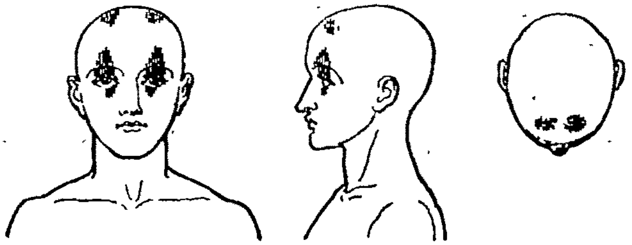


FIG. 2.

To show the areas of superficial tenderness in a case of hypermetropia (Case No. 2). The tenderness marks out the mid-orbital area.

Case 2.—William G. (Mr. Tweedy.)

Complains that he cannot see to read especially with the left eye.

Vision.—Right eye $\frac{5}{60}$ Jæger 6.
Left eye $\frac{3}{60}$ Jæger 14.

He complains of pain "over the eyes." His eyes "ache when he reads."

Superficial tenderness over the areas in fig 2.

As he was doing no work, he was put under atropine, and at the end of a week all headache and tenderness had disappeared.

Right eye.—Vertical + 2·25D.
Horizontal + 3D.
Left eye.—Vertical + 5·75D.
Horizontal + 6D.

Right eye with + 2 D. spherical, and + 0·5D. cylinder axis vertical he saw $\frac{6}{6}$.

Left eye with + 5D. spherical, and + 0·75D. cyclinder axis 45° he saw $\frac{6}{12}$.

He was in every way a typically healthy youth.

Fronto-temporal.

This area rises from the outer part of the eyebrow with a curve towards the middle line. It laps on to the outer part of the upper lid, and takes the whole of the outer half of the lower lid. It rises for about 3 inches (7 cm.) above the external canthus of the eye, and the inward curve brings it in contact with the fronto-nasal area about 1½ inches (4 cm.) from the middle line of the forehead. At the level of the eyebrow its anterior border is about 2 inches (5 cm.) from the middle line of the forehead. Its posterior border is about 3¼ inches (8 cm.) from the centre of the forehead or 3 inches (7·5 cm.) in front of the anterior part of the insertion of the ear. Thus the gap between the two portions of the mid-orbital area is filled approximately by the upper and anterior parts of the fronto-temporal area. Its anterior border abuts on the mid-orbital below, then on the fronto-nasal, and above on the upper or scalp portion of the mid-orbital (fig. 3).

The maximum spot of this area lies about half an inch above the orbital ridge of the frontal bone, and is easily distinguished by lying somewhat above the level of the maxima of the surrounding areas.

The pain associated with this area is said to be "over the forehead," if it is present on both sides, or "over the temple," if it be represented on one side only. If the patient is asked to point with both hands to the position of the headache, the hands assume a very characteristic attitude. The flat of each hand, with the fingers upwards, is placed over the anterior part of the temple so that the ulnar border of the little finger rests just above the outer part of the eyebrow. All the fingers converge to the centre of the forehead, and the appearance presented by the patient is thus very characteristic.

This but rarely occurs alone in disease of the organs of the head (*cf.* fig. 33), but is common as one of the areas of reference from the thoracic viscera. It occurs frequently in iritis, and, in conjunction with the temporal area, in glaucoma.

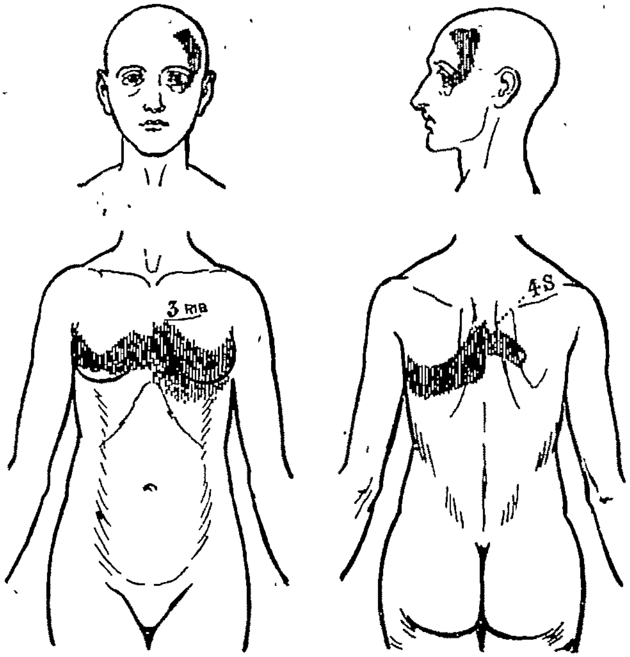


FIG. 8.

To show tenderness of the scalp over the fronto-temporal area. From Case No. 3 (mitral stenosis).

Case 3.—Emily E.[1] (Victoria Park, Dr. H. Sainsbury), aged 36.

Rheumatic fever at 13 years. Second attack at 27 years. Since then "heart has been affected."

Some cyanosis ; pulse 100, regular.

---

[1] I cannot report cases of cardiac and pulmonary disease as fully as I should wish in this section, but can give the salient points only. Most of these cases will be reported in full in my paper on "Mental States in Visceral Disease."

First sound accentuated short and sharp; preceeded by a slight rough murmur; followed by a systolic murmur.

No enlargement of the liver.

No swelling of the feet. No ascites.

Pain over the third and fifth spaces in about nipple line. Also under angle of left scapula. Superficial tenderness over the areas of the fifth and sixth dorsal segments on the left side, and over the fifth dorsal on the right side of the chest.

Headache "over left temple." She places her finger 2½ (6·5 cm.) inches from the middle line over the orbital ridge of the frontal bone. Marked superficial tenderness as in fig. 3.

She remained under observation for six weeks in the hospital, and this form of headache, amongst others, appeared and disappeared several times.

The eyes were normal. No decayed teeth.

Temporal.

This area lies directly over the temporal fossa, and extends upwards for from 3 to 4 inches (7·5 to 10 cm.) above a line joining the external canthus of the eye with the upper part of the insertion of the ear. Its anterior border is about 3 inches (7·5 cm.) from the middle line of the forehead, and about half inch (1 to 2 cm.) in front of a vertical line drawn upwards from the insertion of the ear. Its breadth is about 2 inches (5 cm.) at the widest part (fig. 4).

The maximum lies in the temporal fossa just above the upper border of the zygoma.

The pain is said to be "in the temple," or if bilateral to "shoot through from temple to temple."

This is an exceedingly important area, and appears in affections of the vitreous chamber of the eye (glaucoma), and with caries of certain teeth in the upper jaw. Its great importance arises from the fact that it is associated with the upper gastric area (seventh dorsal) of the chest, and it is therefore peculiarly associated with the presence of nausea and vomiting.

ase 4.—Maria P. (Mr. Tweedy), aged 67.

February 26. At four o'clock yesterday afternoon she was seized with pain in the right eye. Her sight left her and at six p.m., she began to vomit. The vomiting continued till eight a.m., this morning.

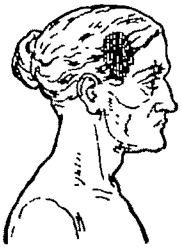


FIG. 4.

From a case of acute glaucoma (Case No. 4). To show superficial tenderness over the temporal area. Some tenderness is present, in a less degree, over the mid-orbital area.

She now has intense pain in the right temporal region, with much slighter pain over the centre of the right eyebrow. The back teeth of the upper jaw ache.

There is marked superficial tenderness over the area in fig. 4.

The cornea is steamy, anterior chamber shallow, pupil of medium size, regular in outline. Tension + 3.

Drops of eserine gr. i. to the ℥j. relieved her greatly.

In the evening Mr. Tweedy did an iridectomy upwards.

February 27. She is now free from pain.

March 18. Wound healed well. All pain and tenderness has been absent since the operation.

The case was a typical one of acute glaucoma.

Vertical.

This area lies exactly over the vertex and abuts in front on the posterior limit of the mid-orbital, fronto-temporal, and temporal areas. Posteriorly it generally just crosses a line drawn vertically from the insertion of one ear to that of the ear on the opposite side. It is roughly triangular in shape, and the apex of the triangle reaches to a point just above and in front of the insertion of the ear. We have now reached that part of the head which varies most in measurement, and it is therefore very difficult to give the limits of this area in inches. Roughly, however, its base along the middle line of the scalp measures about 2 inches (5 cm.). It is best characterised by its relative position to the vertical line from ear to ear, and by the fact that it does not include the extreme anterior portion of the hairy scalp (fig. 5).

The maximum of this area lies just in front of the vertical line joining the ears, and to the right or left of the middle line.

The pain of this area is very definite and is said to be "on the top of the head." The patient puts his hand exactly over the vertical maximum.

This area appears in affections of the extreme posterior portion of the eye and is the hindmost of the eye areas. It also appears with pent-up discharge or rise of tension in the middle ear. It is also associated with hepatic and gastric disturbances, and frequently stands in relation with the well-known "headache on the top of the head" in such disorders.

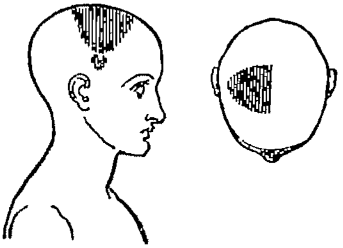


FIG. 5.

To show superficial tenderness over the vertical area of the scalp. From Case No. 5 after excision of the right eye.

Case 5.—Helen B. (Mr. Tweedy), aged 64.

Six months before admission she began to suffer from chronic glaucoma. On admission the right eye was blind. Cornea sodden. Iris dilated. Tension + 3.

She complained of pain around the eye and I therefore excised the right eye, under Mr. Tweedy's guidance.[1]

During the few days she was under observation in the hospital before the operation, no tenderness was present. During the night after the excision she complained of headache on the top of the head, and this headache was associated with distinct superficial tenderness of the scalp (fig. 5). This had entirely disappeared on the following evening.

No pain in the trunk. No areas of tenderness except on the scalp.

---

[1] It is probable that I used more force and produced a greater effect on the nerves than a skilled operator would have done.

Parietal.

This area lies over the parietal eminence. The anterior border corresponds roughly with a line drawn vertically from the insertion of the one ear to that of the other. Its posterior border is roughly formed by a line making an angle of 45° with the vertical line from ear to ear. Thus, if the distance from the occipital protuberance to the root of the nose be from 13½ to 15 inches (34 to 38 cm.), the anterior limit of this area lies from 7 to 8½ inches (about 18 to 22 cm.) behind the root of the nose, and the posterior border about 3¼ to 4 inches (9 to 10 cm.) above the occipital protuberance. It extends downwards towards the upper part of the ear and occasionally seems to involve its extreme upper portion (fig. 6).

Its maximum point lies over the parietal eminence slightly to one side or other of the middle line.

When pain is present over this area the patient places his hand over the parietal eminence.

This area is occasionally affected in diseases of the ear, but never alone. Thus its anterior border can only be determined when its presence is due to disturbance of the lower part of the stomach or some similar visceral lesion.

Case 6.—Hebzibah C. (Victoria Park, Dr. Chaplin), aged 28.

When 1 year old she had measles and whooping cough. This left her with a slight cough. At 8 years old the cough became worse. Became steadily worse each winter, picking up again each summer. At 17 years "bronchitis and inflammation of the lungs." Repeated similar attacks ever since. She has been under Dr. Chaplin's observation for eight years.

Marked signs of fibroid phthisis on the right side. Chest retracted, lateral curvature. Bronchial breathing, bronchophony and large râles over the whole right side from top to bottom.

Respirations 40 in the minute. Pulse 72. Heart drawn completely over to the right side. No murmur.

The whole of the condition described so far is stationary. A few weeks ago she began to suffer from pain in the left side and increased shortness of breath. On admission, besides the stationary signs on the right side, some râles were heard at the left base.

She improved, but then had an increase of signs at the left base with increased shortness of breath.

She then complained of pain in the left side associated with superficial tenderness over the eighth and ninth dorsal areas on the left side.

She also complained of parietal headache associated with the scalp tenderness shown in fig. 6.[1]

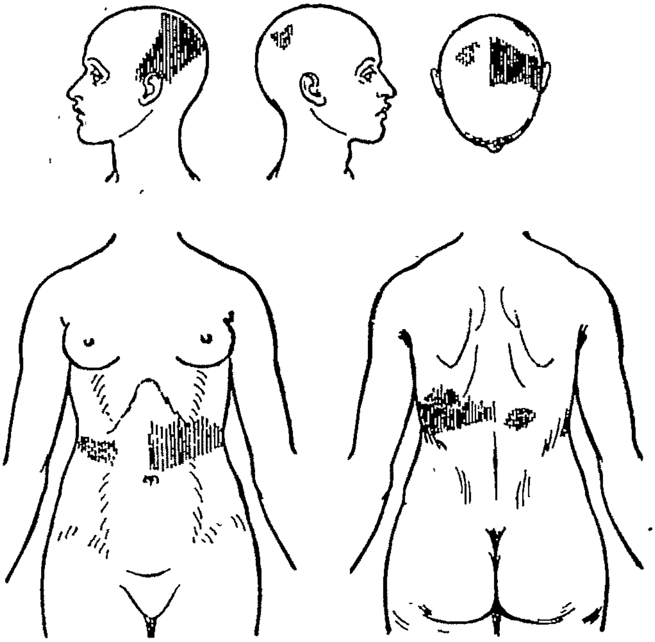


FIG. 6.

To show superficial tenderness over the parietal area of the scalp. On the left side the maximum only is present. From Case No. 6. On the left side of the body the ninth and part of the eighth dorsal areas are affected. On the right side of the body the maxima of the ninth dorsal only were tender.

Occipital.

This area lies in the gap between the posterior border of the parietal area in front of it, and the occipital protuberance. It is roughly oval extending for almost 2 inches (5 cm.) to the right or left of the middle line (fig. 7).

The maximum of this area lies just above the occipital protuberance, and a little to one or other side of the middle line.

---

[1] This case will be fully reported in my paper on the "Mental Changes in Visceral Disease."

When this area is affected the patient complains of pain over the upper part of the occipital protuberance just above the horizontal level of the ears.

Pain over this area is practically always referred from the abdomen excepting in those rare instances where it is associated with lesions of the posterior part of the dorsum of the tongue.

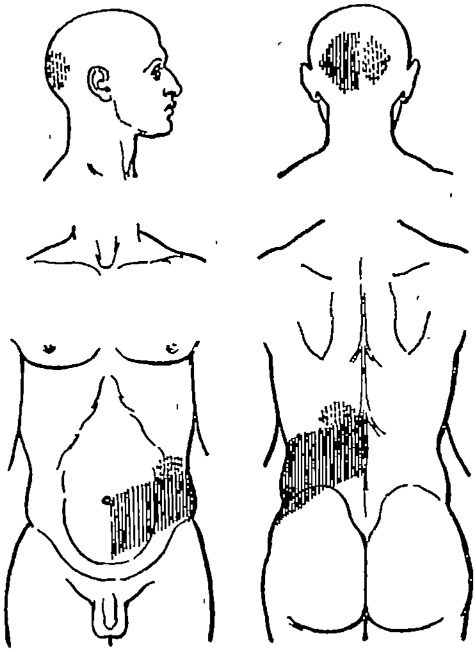


FIG. 7.

To show tenderness of the scalp involving the occipital area (Case No. 7). On the left side of the abdomen and in the left loin the tenth dorsal area is present together with the two maxima of the ninth dorsal area.

Case 7.—Ernest P. (Victoria Park, Dr. Thorowgood), aged 23.

April 21, began to feel very ill, and had pain in the left loin. April 22, he seemed so ill that he was admitted at once.

He is a finely-built, athletic man.

Tongue coated with white fur. Bowels not opened since April 20. No vomiting. Felt sick, April 21 and 22.

No physical signs in abdomen. Spleen not felt. Splenic dulness not increased.

No abnormal physical signs in heart or lungs. Pulse 68, regular, good stroke and tension. Respiration 10 to 12 in the minute. No dyspnœa.

He complained of pain in the left iliac fossa and left loin. Marked superficial tenderness over the area of the tenth dorsal segment on the left side.

Headache "at the back of the head" with superficial tenderness in the occipital region as in fig. 7.

Under the influence of calomel and saline purgatives his bowels became regular, pain and tenderness disappeared, and he was well by April 28.

Rostral.

Seven areas have now been mapped out on the dorsal part of the head. Before passing on to the remaining areas of the face and neck one other area of this group deserves notice. In certain nasal affections we occasionally find that the skin on the front of the nose is tender. Occasionally also in disturbances of the thoracic viscera the bridge of the nose as far as the horizontal level of the alæ nasi seems to be affected. Now the downward flap of the fronto-nasal area does not meet the middle line on the bridge of the nose, but lies more over the lachrymal canal. Thus I cannot help thinking that in front of the fronto-nasal area lies an unimportant, almost rudimentary, patch which I shall call the rostral area.

Further evidence for the existence of this patch will be adduced later when I treat of herpetic eruptions of the face and forehead (p. 376).

B. *Lateral Areas.*

Maxillary.

This is a very easy area to define. It lies over the upper jaw extending as far forwards as the lateral fold between the nose and the cheek. Its upper border is formed by the lower margin of the bony orbit. Its lower border is a curved line joining a point on the cheek, close to the fold between the alæ nasi and the upper lip, with a point just posterior to the bony orbit. The apex of this area lies between the posterior border of the fronto-temporal, and the anterior border of the temporal areas. At the level of the eyebrow the apex lies about 3 inches (7·5 cm.) from the middle line of the forehead (fig. 8).

To this area also belongs that part of the upper jaw which contains the bicuspid teeth, together with a portion of the hard palate. When it is affected from other causes than caries of these teeth (*e.g.*, lesions of the eye) these teeth may ache, and the hard palate in their neighbourhood may be tender.

If this area is affected the patient complains of pain over that part of the cheek which lies over the upper jaw.

This area is sometimes tender with iritis or with rise of tension in the vitreous chamber of the eye. It is, however, most commonly produced by caries of certain teeth of the upper jaw.

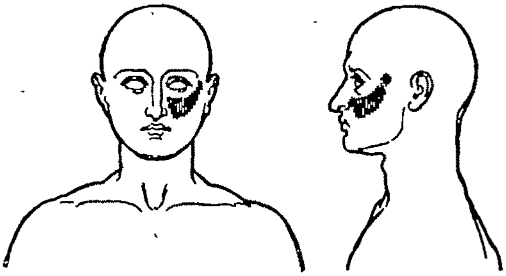


FIG. 8.

To show tenderness over the maxillary area of the face (Case No. 8). The tenderness was due to the first molar tooth of the upper jaw.

Case 8.—Thomas B. (National Dental Hospital).

Three weeks ago he suffered from great pain in the first molar tooth of the upper jaw on the left side. A dentist attempted to extract the tooth, but broke off the crown.

Ever since he has had gnawing pain in the position of the tooth, with pain in the left cheek. The left cheek feels sore whenever he touches it. The roof of the mouth on the affected side also feels sore.

Superficial tenderness over the cheek as in fig. 8.

Both roots can be felt in the socket, and are intensely tender to the point of the probe.

The eye is normal. No pain in the ear or tongue.

Otherwise a perfectly healthy young man. No pain elsewhere in the body.

After extraction the tooth was found to have been broken below the crown. The nerve in the roots was exposed, living and reddened. No periodontitis.

All pain and tenderness disappeared after removal of the tooth.

Mandibular.

This area is also roughly triangular and lies over the coronoid process, the ramus, and part of the body of the lower jaw. Below, it extends as far forwards as a line dropped vertically from the external canthus of the eye. The apex of the triangle lies about ½ inch (1 cm.) in front of the insertion of the ear. From this point the posterior border dips back to include the tragus of the ear, and then swings forwards and downwards in front of the posterior border of the lower jaw. The anterior border of this area abuts on the posterior border of the maxillary area except at its lower portion (fig. 9).

The maximum spot of this area lies about on a level with and a little in front of the tragus of the ear.

The pain of this area is referred to the cheek, just in front of the ear.

This area becomes tender with disease of the last two molars of the upper jaw.

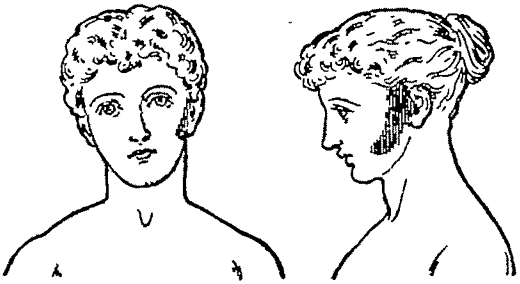


FIG. 9.

To show tenderness over the mandibular area of the face (Case No. 9). The tenderness was due to caries of the second molar tooth of the upper jaw.

Case 9.—Eliza P. (National Dental Hospital), aged 24.

She has suffered from pain, on and off, in the second molar on the left side of the upper jaw.

In addition to the pain in the tooth she has an aching pain in the face, mainly just in front of the ear.

Superficial tenderness as in fig. 9.

No pain in tongue or roof of mouth. No pain in the ear itself, but just in front of the ear.

Not anæmic. Typically healthy, red-faced girl. No pain after food. No vomiting. No pain with menstruation.

The second upper molar on left side reacts to the point of the probe. No other teeth react.

After extraction, the pulp chamber was found to be exposed and there was much sodden dentine. Living pink nerve in each root.

Hyoid.

This area lies partly over the ramus of the lower jaw, and partly behind its posterior border. Its limits are best gathered from fig. 10. Posteriorly it runs up on to the mastoid, and hence superficial tenderness over this area may give rise to fictitious "mastoid tenderness." The lobule of the ear is also tender. The anterior border of this area lies in front of the lobule, but does not include the tragus which belongs to the area in front (mandibular). When this area is affected the meatus, especially the posterior portion, is intensely tender and the insertion of an aural speculum will give much pain.

When this portion of the skin is tender there is also tenderness, as a rule, over the edge of the tongue. This strip on the tongue is about a centimetre in breadth, and comes forwards to within one to one and a half centimetres of the tip. (*Vide* p. 419.)

The maximum spot of this area on the surface of the head lies just behind, and below the angle of the lower jaw. A second most important maximum lies in the external auditory meatus.

The patient complains of pain in the ear ("earache"), and of pain "just behind the jaw." Hence arises the habitual complaint of pain in the ear with affections of the tonsils, side of the tongue, and molar teeth of the lower jaw. It also explains the pain in the ear so commonly met with in diphtheria.

This area is frequently present in affections of the ear, tonsils, tongue and teeth, and it is one that is liable to lead to many errors in diagnosis. I have seen the membrana tympani perforated without relief, when the fault lay in the molars of the lower jaw. (*Vide* p. 417.)

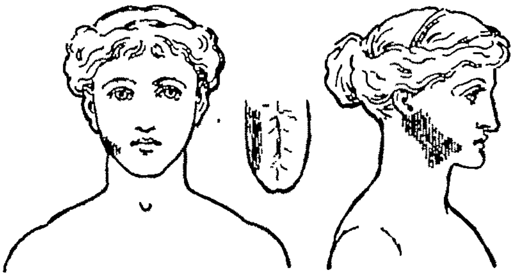


FIG. 10.

To show tenderness over the hyoid area of the face (Case No. 10). The tenderness was due to the first molar tooth of the lower jaw.

Case 10.—Fanny T. (National Dental Hospital), aged 30.

She complains of sharp pain in the ear, and under the right jaw. The back of the jaw aches, and the lobule of the ear has become so tender that she has been obliged to remove the ear-ring. She also complains that the right edge of the tongue is sore.

Definite superficial tenderness as in fig. 10.

The first molar on the right side of the lower jaw reacts to the point of the probe. No other teeth react.

No pain elsewhere in the head and body. No pain after food.

Is not anæmic.

After extraction the pulp cavity was found to be fully exposed. The pulp in the crown was dead, but there was a living reddened nerve in each root. No periodontitis.

A week afterwards she was found to be absolutely free from pain and tenderness.

Superior Laryngeal.

Below the hyoid area we come on to a territory of skin that is exceedingly hard to map out. For the proportions of the front of the neck between the jaw and the sternum vary very greatly. Thus the nature of any area in this region depends on its relation to the surrounding skin rather than on actual measurements in inches from bony points. Moreover the skin of the neck is less sensitive than that of the face to all sensory stimuli.

The area next below the hyoid is roughly triangular in shape. The apex of the triangle reaches posteriorly to the level of a line dropped vertically from the posterior attachment of the ear. The posterior part of its lower border lies just behind the anterior border of the sterno-mastoid, and thence swings downwards and forwards to the level of the lower part of the thyroid cartilage. The upper border runs forwards from the posterior angle, and just laps up on to the extreme posterior border of the body of the lower jaw. It then skirts the fold between the chin and the neck, and seems to meet its fellow of the other side in about the thyro-hyoid space. From this anterior superior angle the base of the triangle curves downwards to meet the termination of the lower border a little to one side of the middle line at the level of the lower border of the thyroid cartilage. Thus, although of smaller size, this area is seen to bear a curious resemblance to the hyoid area in shape (fig. 11).

I pointed out that the hyoid area supplied the sides of the tongue. In the same way I believe the superior laryngeal area supplies the dorsum as far forwards as about 3 cm. from the tip.

The maximum point of this area lies just in front of the anterior border of the sterno-mastoid at the level of the pomum adami.

If this area is affected from any cause whatever the patient complains that he has difficulty in swallowing.

The pain is said to lie " in the throat," and the patient points to the situation of the maximum spot.

Though anatomically a small area, and one that has been crowded in the course of development, it is still functionally important. For into it refer the wisdom teeth of the lower jaw, and the posterior part of the dorsum of the tongue.

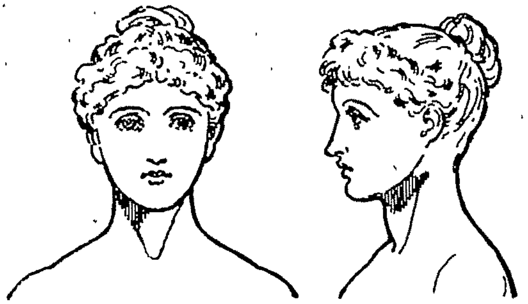


FIG. 11.

To show tenderness over the superior laryngeal area of the neck (Case 11). The tenderness was due to the wisdom tooth of the lower jaw.

Case 11.—Annie B. (National Dental Hospital), aged 20.

She complains of pain in the wisdom tooth on the right side of the lower jaw.

She also complains of pain shooting down the anterior border of the sterno-mastoid "into the throat."

Superficial tenderness as in fig. 11.

No pain elsewhere. No headache. No pain after food.

No pain in the ear.

The wisdom tooth reacts to the point of the probe. No other teeth react.

After extraction the pulp was found to be exposed by a cavity infiltrating the whole crown of the tooth. Pulp reddened, but living. No periodontitis.

Inferior Laryngeal.

In this area the tendency to elongation downwards, which was manifest in the superior laryngeal, becomes still more striking. The superior angle lies on the anterior border of the sterno-mastoid at about the level of the pomum adami. The upper border runs with a slight curve almost directly inwards to reach the middle line at about the crico-thyroid membrane. The posterior border runs along the anterior border of the sterno-mastoid to the sterno-clavicular articulation. It then bends a little outwards, forming a small patch over the sterno-clavicular joint. The base of the triangle is formed by the middle line (fig. 12).

The maximum point of this area lies over the anterior border of the sterno-mastoid muscle a little below the level of the cricoid cartilage.

This area becomes tender in diseases of the larynx, especially such as affect the vocal cords and parts below them. The pain is said to be situated over the anterior border of the sterno-mastoid at about the level of the cricoid cartilage. If this area is tender the patient complains that he has a feeling as if there was something in his throat, and thus the so-called "globus," in one or another form, may be a symptom of grave organic disease of the larynx.

Moreover, stimulation over this tender area will cause a feeling of tickling in the throat and thus frequently produce a reflex cough.

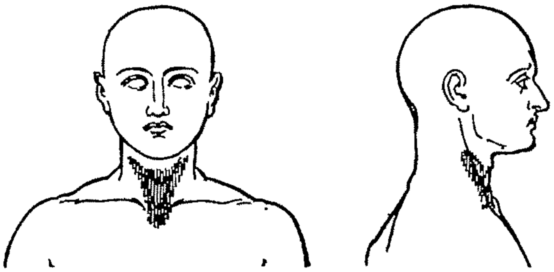


Fig. 12.

To show tenderness over the inferior laryngeal areas of the neck (Case No. 12). The tenderness was due to ulceration of the larynx.

Case 12.—George G. (Victoria Park, Dr. Thorowgood), aged 42.

As a young man he suffered from cough and once spat blood. At 30 he had another attack in connection with a "cold." Sweated at night and wasted. Recovered and remained fairly well till November, 1892. He then "caught a cold," had a troublesome cough, and two weeks later lost his voice. Has been ill ever since, recovering a little with the warm weather, but becoming worse again with the winter.

October, 1893.—*Condition of Larynx.*—Epiglottis reddened, slightly swelled and quite immovable. Arytenoid mucous membrane swollen and œdematous. On the right arytenoid is a superficial ulcer. Left ventricular band ulcerated longitudinally, with a yellowish slough.

He complains of pain and soreness over the neck between the anterior borders of the two sterno-mastoids.

Marked superficial tenderness over the areas in fig. 12.

There were signs of consolidation at both apices and at the left base, but throughout the two months he was under observation the destructive changes in the lungs advanced very slowly.

On the other hand the destruction of the larynx advanced rapidly, and he repeatedly suffered from pain in the throat associated with cutaneous tenderness over the superior and inferior laryngeal areas. I have selected an occasion on which the inferior laryngeal area was mainly affected. Whenever this area of tenderness was present a cough could be excited by stimulating the tender skin.

Died November 30.

Post-mortem.—The larynx was much ulcerated. The base of the epiglottis was thickened and ulcerated. The whole of the left cord was practically destroyed. The right was ulcerated but not to the same extent, and appeared reddened even after death. The arytenoid cartilages were ulcerated and nearly destroyed. There was an ulcer in the trachea.

It was a typical case of tubercular ulceration of the larynx.

This is the lowest of the areas into which reference takes place directly from organs in the head or neck.

But before passing on to the two areas on the lateral aspect of the neck, I must return to two areas on the face which I have so far neglected.

C. *Anterior Areas of the Face.*

Naso-labial.

This area lies over the upper lip, tip and under surface of of the nose, and on the cheek. It includes part of the lower lip and extends, as a patch on the cheek, as far back as a line dropped vertically from the external canthus of the eye. It is exceedingly difficult to describe in words, and can best be understood from fig. 13. It is not easy to mark out the limits of this area except in some cases of caries of the teeth.

When present, the patient complains that his upper lip is "swollen and tender" and that the "tip of the nose is sore."

This area is associated with lesions of the respiratory portions of the nose, but under such circumstances is mostly accompanied by the rostral, and frequently by the fronto-nasal areas. With caries of the teeth it may, however, appear alone.

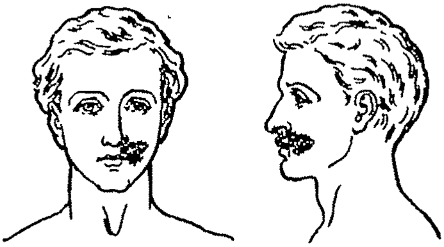


FIG. 13.

To show tenderness over the naso-labial area of the face (Case No. 13). The tenderness was due to the canine of the upper jaw.

Case 13.—Stephen R. (National Dental Hospital), aged 21.

He complains of pain in the left upper canine and of pain in the face a little behind and above the level of the angle of the mouth.

Superficial tenderness as in fig. 13.

The tongue is not sore. Roof of mouth not sore.

Left upper canine reacts to the point of the probe. No other teeth react.

The canine was drilled and a living nerve was killed with an arsenical dressing. Later the tooth was stopped and all pain disappeared.

Mental.

This area swings almost directly back from the angle of the mouth to a point in a line drawn vertically downwards from the external canthus of the eye. Thence it drops slowly to a point on the posterior border of the body of the lower jaw, about 2½ to 3 inches (6 to 7·5 cm.) from the middle line. It then runs forwards along the fold separating the chin from the neck. In front it does not include the lower lip or that part above the elevation of the chin (fig. 14).

When this area is affected the anterior 3 cm. of the tongue are tender, on the side affected.

The maximum point of this area lies close to a vertical line dropped from the angle of the mouth.

The pain is said to be situated over a point on the lower jaw close to a vertical line drawn downwards from the angle of the mouth. This point lies close to the point where the mental nerve enters the superficial structures from below.

This area is tender in connection with disease of the anterior part of the tongue and of the incisor and canine teeth of the lower jaw.

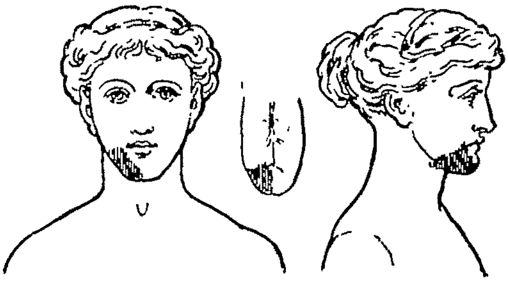


FIG. 14.

To show tenderness over the mental area of the face (Case No. 14). The tenderness was due to the canine of the lower jaw.

Case 14.—Ada C. (National Dental Hospital), aged 30.

For ten days she has suffered from pain in front of the lower jaw. She points to a spot just below the angle of the mouth on the right side. The tip of the tongue is sore. Pain in right lower canine.

Marked tenderness of the face and of the right side of the tip of the tongue as in fig. 14.

The canine on the right side of the lower jaw reacts to the probe.

No pain elsewhere in body or head. Tongue normal, excepting for the tenderness of the right side of the tip, for which there is no obvious cause in the tongue itself.

After extraction the root was found to contain a living reddened nerve. There was a mass of sodden dentine over the exposure. No periodontitis.

Thus I have now described a series of dorsal areas, a series of lateral areas, and two areas on the anterior aspect of the face. Below the mental area there are no anterior areas, and the homologous portions in the neck seem to have doubled inwards and to appear on the tongue.

D. *The Areas of Neck.*

Sterno-mastoid.

The upper portion of this area lies behind the ear and over the insertion of the sterno-mastoid muscle, into the occipital bone, at the superior curved line. The descending portion of the area lies over the sterno-mastoid and almost exactly follows the anterior border of this muscle. On the front of the chest it reaches the upper border of the second rib. It covers the inner third of the clavicle and the supra-clavicular fossa. The posterior border of this area swings away from the occiput, rapidly joining the anterior border of the upper part of the trapezius (cucullaris) muscle to end on the shoulder close to the inner and anterior aspect of the shoulder joint. The lower border then rises again to a point above the clavicle in the outer part of the supra-clavicular fossa. In this way a notch is formed between the anterior angle, which is pulled down on to the chest, and the posterior angle, which is pulled down over the outer part of the clavicle. This area can clinically be quickly differentiated from the next area below it by its large basi-occipital flap and from the fact that except for this extreme upper part none of its territory can be seen from behind (fig. 15).

One well-marked maximum lies over the hollow between the insertion of the trapezius (cucullaris) and of the sterno-mastoid into the skull. A second maximum lies over the supra-clavicular fossa.

The pain of this area is said to be an aching "over the neck and back of the head" or is called "a stiff neck."

This area is not primarily tender in disease of any organ of the head or neck. But it is a very important area in connection with pain due to disturbances of organs in the chest.

Case 15.—Joseph C. (Victoria Park, Dr. Lyon), aged 30.

Was quite well till three years ago, when he "caught cold after cold." Since then has never been quite free from cough. Sputum occasionally tinged with blood; not profuse. Has sweated at night for two years. Has been wasting for seven to eight weeks.

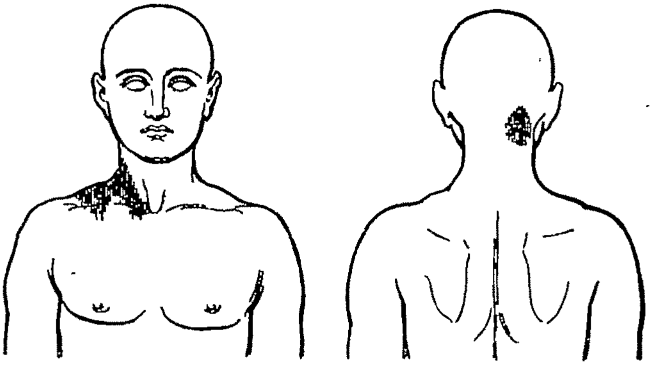


FIG. 15.

To show tenderness over the sterno-mastoid area of the neck (Case No. 15). The tenderness was due to tubercular disease at the apex of the right lung (acute outburst).

September 18.—Condition as follows :—

Cough not very troublesome. Respiration 36. Short of breath on exertion. Sputum muco-purulent, not profuse ; no blood.

Diminished movement, and higher pitched note under right clavicle. · Over right apex expiration prolonged with whispered bronchophony. Behind, over right apex, diminished percussion note; expiration prolonged. Fine crackling râles heard over right apex both back and front, and in supra-clavicular fossa.

Over the left side percussion note was good. Breath sounds possibly a little weak over left apex. A few crackling râles heard.

Pulse 88. Heart sounds normal.

He was free from pain and tenderness.

He improved somewhat ; gained 2lb. Temperature ranged from 100° F. (37·8° C.) at night to 98° (36·7° C.) in the morning.

On November 15, he woke with a sharp pain over the right shoulder. (Examination of the right shoulder revealed no physical abnormality.)

The next day he complained of an aching feeling "as if he was bruised" over the right supra-clavicular fossa, and right side of the neck. This lasted for a week, and was associated with the superficial tenderness shown in fig. 15. Sometimes this tenderness diminished to a spot, sometimes it was present as a full area.

On November 15 and 16 the temperature did not fall below 99° F. (37·2° C.), and rose on the 16th to 101·2° F. (38·5° C.) He began to sweat profusely and ceased gaining weight.

From this time râles were heard over the right back as low as the angle of the scapula, and the signs at the right apex in front became more active.

When he left the hospital on December 10 he had lost 2½lb. His temperature was ranging from 101° F. (38·3° C.) to 98° F. (36·6° C.) in the 24 hours. His shoulder was still painful at times. The signs were evidently increasing over the upper part of the right lung. The left apex was quiet.

Thus I cannot help thinking that the pain and tenderness shown in fig. 15 were due to a sudden increase in the activity of the destructive changes at the right apex dating from November 15.

Sterno-nuchal.

The dorsal border of this area occupies from about the fourth to the seventh cervical spines. From about the seventh cervical spine, its lower border runs outwards and downwards to the spine of the scapula and covers the upper part of the trapezius (cucullaris) muscle. It sends a flap downwards over the posterior aspect of the shoulder joint and then swings over the shoulder, in the region of the acromion. From the tip of the acromion it dips sharply downwards into the groove between the deltoid and the clavicular portion of the pectoralis. It descends on the arm to the level of the lower border of the pectoralis, and then rises to the front of the shoulder joint, to end on the front of the chest over the third rib (fig. 16).

The pain of this area is said to lie over the shoulder joint, both in front and behind, and occasionally pain is produced when the patient moves his arm on the shoulder. No pain is however produced by gentle passive movement at the shoulder joint.

This area of tenderness appears solely in answer to disturbances in the thorax and abdomen, and is not affected by lesions of any organ of the head and neck.

Case 16.—James C. (Victoria Park, Dr. Thorowgood), aged 43, sawdust dealer.

He has had many accidents in the course of his work.

June, 1893.—Became short of breath with a slight cough, went to Guy's Hospital where clear fluid was removed from his chest.

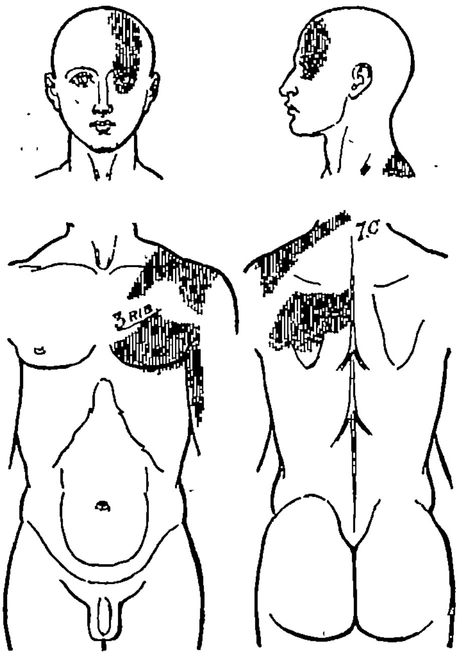


FIG. 16.

To show tenderness over the sterno-nuchal area of the neck (Case No. 16). This fig. also shows tenderness over the third and fourth dorsal areas on the chest and over the mid-orbital and fronto-temporal areas of the scalp on the left side (chap. iii.). Compare with this, fig. 45, p. 482.

On discharge felt well until he tried to work. He then found that the pains in the left side, of which he had complained on and off for some time before admission to the hospital, were as bad as ever.

Alcohol four to six half-pints daily. Denies gonorrhœa and syphilis. Married.

On admission (*December*, 1893), he was well nourished. Largely built man. Very intelligent.

Respiration 17. Slight single occasional cough.

Some pain above left nipple darting through to scapula behind. Definite superficial tenderness, which I shall describe in a subsequent paper.[1]

Pulse 76, regular. The right pulse is stronger than the left all up the arm.

Apex beat fifth space one inch outside nipple line. No pulsation elsewhere in the chest. No thrill; suspicion of dulness on percussion over upper part of sternum at level of second rib. Cardiac dulness otherwise a little diminished. Loud systolic murmur over upper part of sternum and second right intercostal space sometimes conducted to apex, but not heard in the axilla. Murmur heard in arteries of the neck. No diastolic murmur, second sound heard over second right interspace; second sound over apex beat good, first sound sharp, full toned.

---

[1] Part iii. of this investigation.

Signs of an old pleurisy at the right base. Slight emphysema but otherwise no abnormal signs in the lungs. Larynx normal.

Tongue clean. Bowels confined. Appetite good; no pain after food. Liver not enlarged.

Whilst in the hospital he gradually lost the pain and tenderness, put on weight, and went out January 20, much improved.

He began to work at the end of March, 1894, and I saw him on April 10, with all the pain and tenderness back again over the areas in fig. 16.

Physical signs in the chest exactly as before. Pulse 80, small stroke, not regurgitant. Signs at the base of the right lung exactly as before. Nothing in the abdomen.

The sterno-nuchal is the last of the areas on the neck which are tender from visceral disease. The next area below, that appears in consequence of visceral disease, is the dorso-ulnar or area of the first dorsal segment. Thus, as I showed in my first paper, the upper gap, into which no direct reference takes place, lies between the sterno-nuchal above and the first dorsal area below.

§ 2. *Herpetic Eruptions of the Head and Neck.*

When discussing the areas of tenderness on the body I pointed out that they resembled the distribution of herpes zoster. As each tender area was described, an instance was given to show the corresponding distribution of an herpetic eruption. Thus zoster formed an interesting corroboration of the existence of areas having the likeness and form of the hyperalgesic zones I had described.

On the neck and shoulders herpes still follows the lines laid down in the first half of this chapter. On the forehead and scalp I do not think that these lines are transgressed, but there is a greater tendency for the areas to appear in groups than is the case on the body. On the face this tendency is exaggerated, and in many cases it would appear at first sight as if the lines laid down from the study of the tenderness in visceral disease were set at naught. I shall, however, try and show that, if a considerable number of cases are examined and carefully drawn, some order seems to appear in the apparent chaos.

The results detailed in this section are based on 78 cases of herpes on the body and limbs, 19 cases on the neck and shoulder, 11 cases on the scalp, and 45 selected cases on the face, collected and drawn by myself.

Herpes of the Scalp and Forehead.

The fronto-nasal area is not uncommonly mapped out by herpes, but it mostly occurs with one of its neighbours. Fig. 17 shows this area in a case where the nasal portion was not covered by the eruption but was intensely tender. When this area is affected by herpes a shallow ulcer is very liable to appear on the cornea.

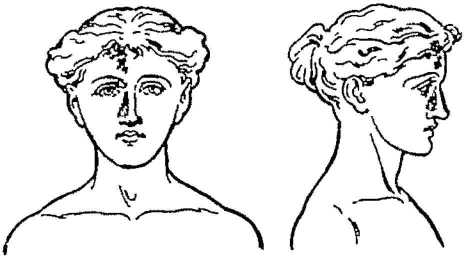


FIG. 17.

To show the distribution of an herpetic eruption over the upper part of the fronto-nasal area (Case No. 17). The dotted area on the forehead represents the eruption. The shaded area on the nose was intensely tender but was not covered with vesicles (*cf.* fig. 1).

Case 17.—Annie R., aged 20. October, 1891.

Three days ago eruption began to come out, and "the forehead ached like fire;" last night upper eyelid swelled. Nose swelled and became tender.

The eruption follows the area in fig. 17. There is great tenderness to the head of a pin over the shaded areas on the nose.

Slight conjunctivitis. Eye has watered for the last two days. No ulcer on cornea.

Last lower molar decayed. No other bad teeth.

The next figure (fig. 18) shows an eruption that occupied the mid-orbital and fronto-temporal areas in addition to those in front. The posterior limit of the eruption was 2¼ inches (5·5 cm.) from the vertical line joining one ear with the other.

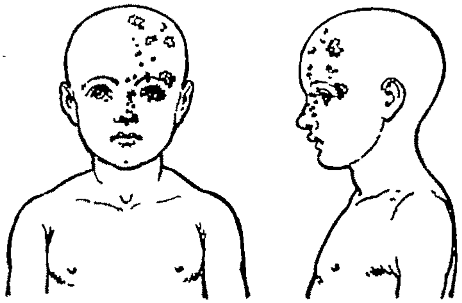


FIG. 18.

To show an herpetic eruption occupying the rostral, fronto-nasal, mid-orbital and fronto-temporal areas (Case No. 18).

ase 18.—Hannah P., aged 6. January, 1893.

January 8. She was a little hot and thirsty. Thought to be "feverish," and was given a "cooling powder."

January 9. A few spots appeared over the forehead, just below the border of the hair.

January 10. The left eye began to swell, and the eruption came out on the temple. She vomited two or three times in the morning and again in the afternoon.

January 11. Admitted to the eye ward under Mr. Tweedy. Weakly looking child.

Very marked herpes of the forehead; the vesicles are large, and several of them have raised, inflamed edges. The largest is situated on the outer part of the eyebrow. There is some swelling and œdema of the anterior part of the scalp. Both lids much swollen and œdematous. A large vesicle is situated over the margin of the upper lid at about its centre, and overlaps on to the conjunctival surface.

Conjunctiva reddened and injected. Cornea clear. No ulcer. Iris clear, dilates well under atropine.

The gland close to the lobule of the left ear is enlarged.

January 14. A tiny roughened pit is present on the outer edge of the left corneo-scleral margin.

Occasionally the complete distribution of the first or ophthalmic division of the fifth may be marked out by herpes. I give a case (fig. 19) in which the eruption closely corresponded to the distribution of the ophthalmic division of the fifth as determined by Sherrington and myself. (*Vide* chap. v.)

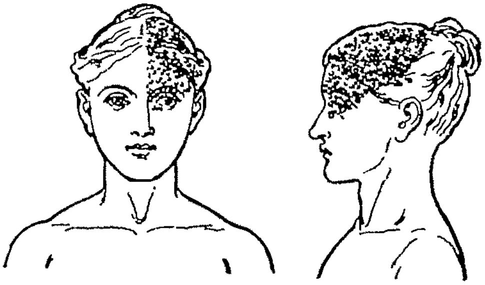


FIG. 19.

To show an herpetic eruption occupying the greater part of the fronto-nasal, mid-orbital, fronto-temporal, temporal and vertical areas (Case No. 19). The distribution of this eruption closely resembles the supply of the superior (ophthalmic) division of the fifth nerve.

Case 19.—Hannah D. (Royal Free Hospital, Dr. Sainsbury), aged 43.

October 22. She began to suffer from pain up the nose, over the left eyelid and on left side of scalp. "As if very strong sticking-plaster were dragging the flesh from the bone."

October 24. Two small spots came out on upper part of forehead.

October 26. Eruption came out all over the area in fig 19.

October 27. She was admitted into hospital. Well-marked herpetic vesicles over the area in the figure.

Chemosis. Tiny elevated spot at outer corneo-scleral border. Lower lid much swollen.

Distinct slight iritis.

Left optic disc red and injected, like that frequently seen with hypermetropia. Inner and lower edge cannot be clearly defined.

Right disc normal. Differs markedly from the left disc. No error of refraction in either eye.

Temperature.—October 27: Morning, 98·4° F. (36·9° C.); evening, 101° F. (38·3° C.). October 28: Morning, 98·2°; evening, 100·8°. October 29: Morning, 98·°; evening, 98·8. After this the temperature did not rise above normal.

It will be remembered that when speaking of the areas of hyperalgesia on the nose I pointed out that if the fronto-nasal area be drawn on both sides of the forehead, a portion of the nose remains unaffected between the two descending nasal limbs. Now this area is never affected alone in any case of referred pain and tenderness. But in some cases of disease of the nose, and in certain cases of tenderness associated with disturbances of the thoracic viscera, this gap seems to be filled in. I was therefore forced to conclude that this patch on the front of the nose formed the extreme anterior portion of the dorsal areas of the scalp.

Now in many cases of frontal herpes the whole of the nose to the middle line is covered by the eruption excepting over the ala nasi, and under surface of the tip (fig. 18). Thus here again we see signs of the presence of a rostral area.

I have no case showing the temporal area alone affected, but I give one (fig. 20) in which this area appeared with herpes around the upper lip.

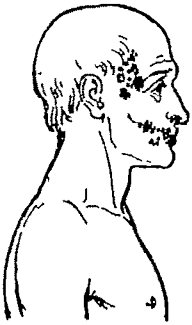


FIG. 20.

To show an herpetic eruption over the temporal and naso-labial areas (Case No. 20).

Case 20.—George M. (U.C.H., Mr. Barker), aged 55. He was admitted November 1, 1892, for urethro-rectal fistula. Bougie passed occasionally.

November 13. No. 7 French bougie passed without difficulty. Followed by rigor. Temperature rose to 102·8 F. (39·3° C.). In the evening sudden pain in left leg below the knee. No pulsation felt in dorsalis pedis, posterior tibial or popliteal arteries of left leg.

November 17. Herpes came out over the temporal region and around the mouth (fig. 20).

This case is published in full by Mr. Barker in the *Bristol Med. Chir. Journal*, June, 1893.

Herpes of the Neck and Arm.

Those forms of herpes which follow the areas on the front of the neck are mostly double. Fig. 21 shows the superior laryngeal area on the left side, and the superior and inferior laryngeal areas on the right side.

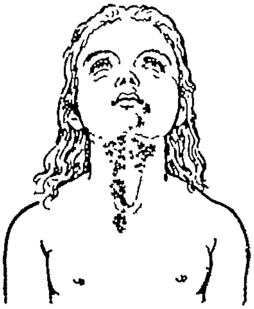


FIG. 21.

To show the distribution of the herpetic eruption in Case No. 21. On the right side the eruption occupied the superior and inferior laryngeal areas; on the left side the superior laryngeal area was affected (*cf.* figs. 11 and 12).

Case 21.—Rose C., aged 6.

June 9 she seemed ill. She complained that her throat was very sore and ate hardly anything. She was very hot at night.

June 10 vomited. Still took nothing to eat. Complaining that her throat was sore. At three p.m. the eruption began to come out.

June 15. The eruption follows the lines in fig 21.

She has a curious cough somewhat resembling whooping cough. Signs of bronchitis and emphysema in the chest.

The next area is the sterno-mastoid, shown in fig. 22. All its characteristic points are well marked; the large sub-occipital flap, the outer part over the shoulder, and the inner part over the supra-clavicular fossa are all well shown. It is noteworthy that this area always sends a small anterior portion to a point on the cheek just below the ear. This anterior flap is too small to be recognised clinically in the corresponding area of tenderness, but I cannot help thinking that I have more than once detected signs of its presence in cases of phthisis.

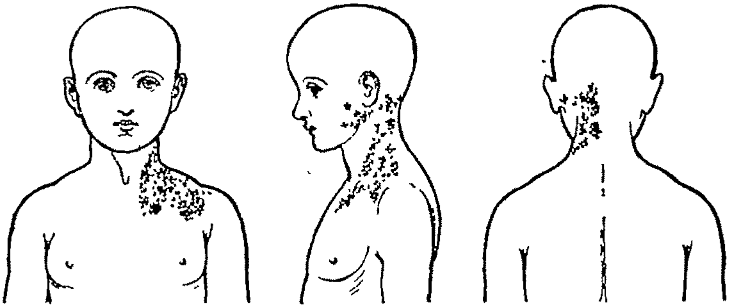


FIG. 22.

To show an herpetic eruption occupying the sterno-mastoid area (Case No. 22). Compare fig. 15 and fig. 58.

Case 22.—Daisy B., aged 6, October 24, 1891.

For some days she has been complaining of pain extending from the left ear into the neck. To-day the first spot of herpes came out over the first intercostal space and second rib.

October 25, the spot in the supra-clavicular fossa appeared.

October 27, the eruption came out fully.

No cough. No signs in lungs. Appetite good. Bowels open daily.

Next below the sterno-mastoid comes the sterno-nuchal area (fig. 23).

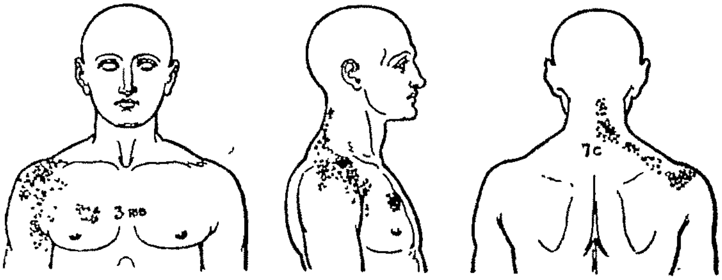


FIG. 23.

To show an herpetic eruption occupying the sterno-nuchal area (Case No. 23). Compare fig. 16.

Case 23.—Francis W., aged 20.

June 21, 1891. He felt a burning pain in his left shoulder, and the eruption began to come out over the posterior aspect of the shoulder joint. The advent of the eruption was not preceded by pain for any length of time.

The spot on the chest came out on June 24.

No cough. No physical signs in the chest. Bowels regular. Appetite good.

These two areas are particularly prone to occur together and occasionally the eruption occupies in addition the superior and inferior laryngeal areas.

We have now reached the limit of the areas over which tenderness is present with referred pain. Thus the upper gap, occupying the greater part of the arm, would be *terra incognita* but for herpetic eruptions. For whilst the lower gap, situated over the leg, could be approximately filled in by the study of organic lesions of the spinal cord, such lesions are, owing to their fatal nature, too rare to enable us to fill in the gap on the arm.

Herpes of the upper limb is rare and frequently imperfect. But in the course of three years I have succeeded in collecting an almost complete series of cases. The one missing area in my series is supplied by a case reported by Pfeiffer. By a complete series I mean cases in which what I take to be one area has appeared alone. Thus I assume that many of the remaining cases in my possession represent eruptions that occupied more than one area and it may turn out on further study that the cases I have selected as representing single areas in reality are more or less incomplete eruptions. This time alone can decide.

The first area below the sterno-nuchal is still attached to the spine, but the lateral portion is much pulled out so that it reaches the front of the elbow on the radial side. It no longer runs up to the middle line of the chest (fig. 24).

Case 24.—Sophia M., aged 49.

The eruption came out October 19, 1891. She had no pain except when the eruption was actually coming out. Since it has come out she has had an aching pain over the deltoid. It hurts her to raise her arm.

The extent of the eruption is shown in fig. 24.

No pain on breathing or coughing. No signs in chest. No cough. Appetite fair. Bowels opened daily.

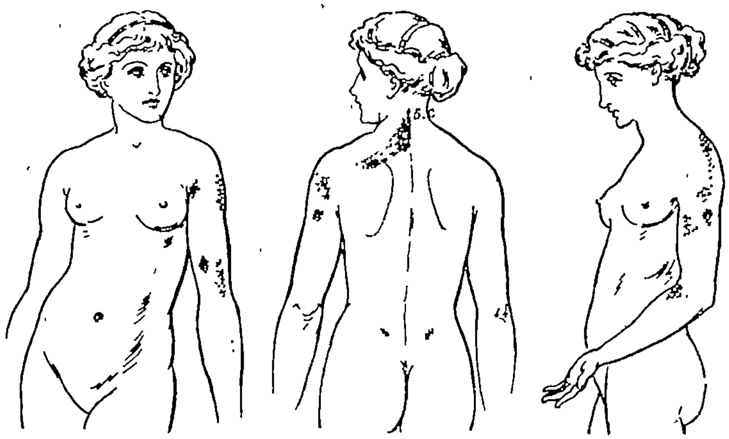


Fig. 24.

To show an herpetic eruption occupying the area of the fifth cervical segment (Case No. 24). This shows the first area of the upper gap.

The second area of the gap is still attached to the spine, but its anterior limb runs down the arm and forearm on the anterior surface to a point just above the wrist. The anterior angle of the first area of the gap just reached the anterior axillary fold. But the anterior angle of the second area lies to the radial side of the anterior axillary fold (fig. 25).

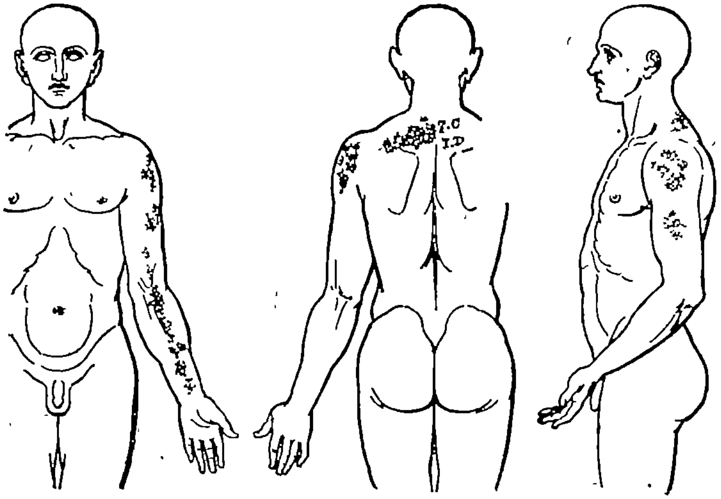


Fig. 25.

To show an herpetic eruption occupying the area of the sixth cervical segment (Case No. 25). This shows the second area of the upper gap.

Case 25.—Mr. Y., aged 21. Student of law. (Dr. Edmonds.)

December 9, he was feverish, hot, thirsty. Lassitude and disinclination to work.

December 10. Eruption came out.

He had no pain until just before the eruption came out (fig. 25).

No vomiting. No cough. No signs in chest. Bowels opened daily.

The third area has lost all connection with the spine behind or with the chest in front and occupies the radial side of the forearm anteriorly. If the arm is straightened and the hand placed with the ulnar border against the body and the thumb turned out this area almost covers that part of the arm seen in side view. On the hand it occupies the thumb and thenar eminence, in front and posteriorly the thumb and index finger (fig. 26).

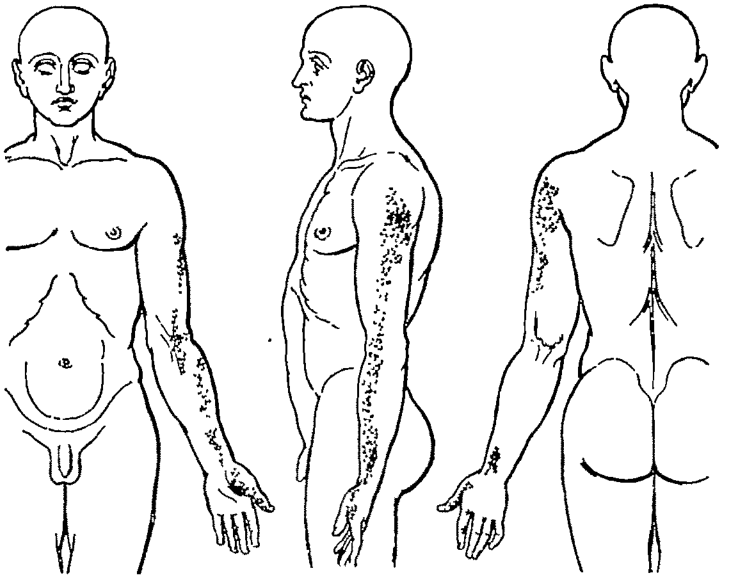


FIG. 26.

To show an herpetic eruption occupying the area of the seventh cervical segment (Case No. 26). This shows the third area of the upper gap.

Case 26.—Charles Henry E., aged 13.

May 10, 1893, he began to complain of pain down the radial side of the arm.

May 14, he became much worse. Vomited. Had been "hot and feverish" for a day or two, but this became worse and it was thought that he was sickening for the measles. He complained that he could not move his arm. He held it by the side and would not use it for anything.

May 17. Eruption came out (fig 26). The spot on the thenar eminence is covered with a large vesicle containing black fluid ("blood blister"). The top of the thumb is covered with confluent vesicles containing clear fluid.

He suffers from chronic cough. Slight diminution of percussion note over right apex behind. Bronchial breathing, increased vocal resonance and a few râles heard over right apex.

No obvious weakness of right arm. No paralysis of any muscle or movement. He complains, however, that movement is painful.

The fourth area lies mainly on the back of the arm and on the hand. It lies more to the ulnar side of the arm than the area I have just described. On the front of the forearm it swings from the radial side of the elbow to the ulnar side,

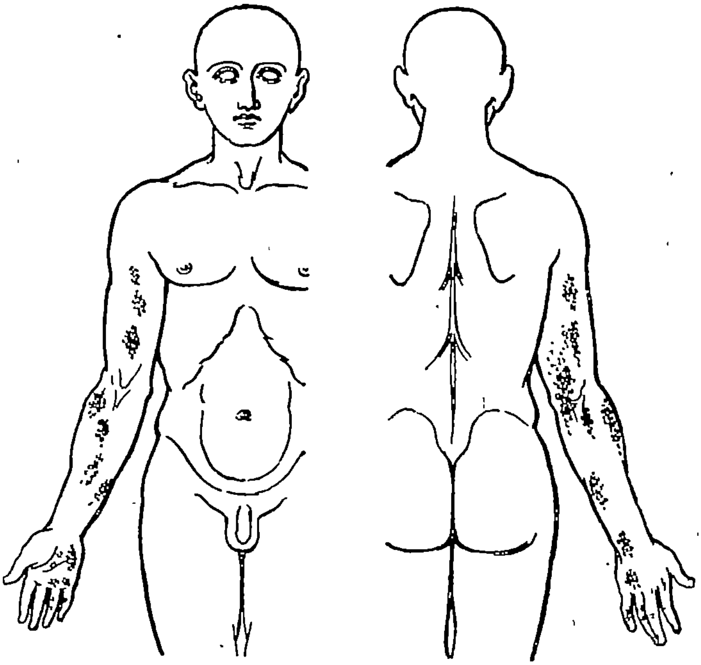


FIG. 27.

To show an eruption occupying what I believe to be the area of the eighth cervical segment. (Reproduced from Pfeiffer, *op. cit.*)

leaving the extreme ulnar border of the forearm and hand free. It occupies the palm of the hand, the ulnar side of the back of the hand and the whole of the ring finger. I believe it also affects the palmar surface of the index and middle fingers and the terminal two joints of the little finger.

I possess no pure case in my collection showing this area, though I have seen it in combination with the two areas below. I therefore give a reproduction of a figure by Pfeiffer.[1] Von Baerensprung[2] also figures a case which much resembles this area, although it seems to have extended somewhat further up the arm.

The next area below this is the highest of the series I described in my first paper. (Fig. 28 is a reproduction of fig. 12 in my first paper.)

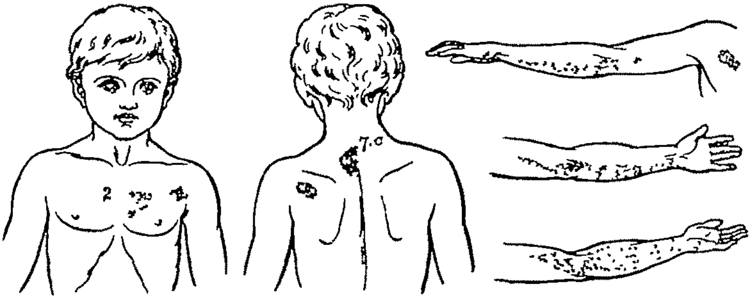


FIG. 28.

This is a reproduction of fig. 12 (Case No. 18) from my first paper (BRAIN, 1893, p. 33). It shows an herpetic eruption following the distribution of the first dorsal segment.

Now, if we accept the nomenclature I adopted in that paper,[3] this area belongs to the first dorsal segment. We can, therefore, count back as follows:—The fourth area of the gap (fig. 27) will represent the eighth cervical segment. The third area (fig. 26) will be the seventh cervical, the second the sixth cervical, and the first area of the gap will represent the fifth cervical segment of the cord. This makes the sterno-

---

[1] "Die Verbreitung des Herpes Zoster," Jena, 1889, Tafel vii.

[2] *Annalen des Charité Krankenhauses*, Bd. x., Heft 1, 1862, Tafel i.

[3] Thorburn's criticism of this nomenclature will be dealt with in Part ii.

nuchal the fourth cervical, and the sterno-mastoid the third cervical segmental areas of the cord.

Thus the gap on the arm appears to lie between the fourth cervical and the first dorsal segments.

Herpes of the Face.

The herpetic eruption on the lips that accompanies a cold or a febrile attack consists of small papules, which form scabs and fall off. It may occur time after time in the same position, is never widely spread, and in the character of its lesions does not resemble herpes zoster. If, however, a large number of cases of hereptic eruptions on the face are examined, we occasionally find cases in which the eruption assumes the typical vesicular form seen on the body. There are also some intermediate forms in which one portion may be truly vesicular and another portion may not have advanced beyond the papular stage. Now, whereas it is the rule to find that a patient has suffered several times from the "febrile" form (papular) of herpes, it is much less common (in my experience very uncommon) to find that a patient has had two attacks of the true vesicular form on the face.

Fig. 29 shows one of these cases in which the eruption was truly vesicular. The site of the eruption is that not uncommonly occupied by the papular form, but in this case the eruption consisted of large, clear vesicles on a reddened base, exactly like those on the body.

The resemblance that the distribution of this eruption bears to the tender area (naso-labial) shown in fig. 13 is noticeable.

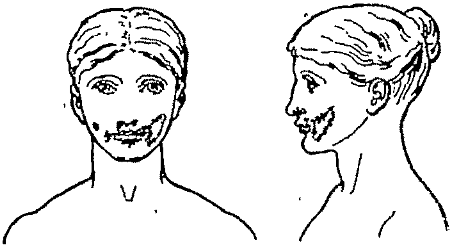


FIG. 29.

To show a true vesicular herpetic eruption occupying the naso-labial area of the face (Case No. 27). Compare fig. 13.

Case 27.—Mrs. W., aged 34.

January 21, 1892, she felt ill, shivering and cold, with aching pain in all her limbs. She has had a cough for some time, but it now became worse.

On the evening of January 23 she felt a burning and itching about the lips and around the mouth. In the night the eruption came out over the area in fig. 29. It consists of large and very marked vesicles on a reddened base, very different from the eruption usually seen around the lips. The tongue is not affected. No signs in lungs. Baby is one month old, and she is suckling it. No vomiting. Bowels confined for two days.

Another form of herpes on the face tends to affect the tip of the tongue, and resembles the mental area (fig. 14) in its distribution.

Thus, though many cases of so-called herpes of the lips do not resemble zoster, I cannot help thinking that the lips and lower jaw may be the seat of an eruption exactly analogous to zoster on the body or neck.

On the face herpes tends to follow curious lines. Thus the naso-labial, mental, superior and inferior laryngeal tend to appear in couples. The commonest arrangement is the naso-labial with the mental or the superior with the inferior laryngeal; but the superior laryngeal may be coupled with the mental. If two of these areas are affected, those which lie next one another tend to appear together, and I do not think the naso-labial appears with the superior or inferior laryngeal without the intervening mental area. In the same way the maxillary mandibular and hyoid areas may appear together.

Another form of combination is for the temporal area to appear with the maxillary or naso-labial areas; or for the mandibular to appear with the mental area; again the occipital may appear with the superior laryngeal.

But beyond these rough rules it is very difficult to foretell the distribution of a herpetic eruption on the face. There seems to be an extreme tendency to spread over the whole of that part of the face made up of the maxillary, naso-labial, mandibular, mental and temporal areas.

§ 3. *The form assumed by the Cervical Areas.*

The typical distribution of a spinal segment to the skin takes the form of a band running from the mid-dorsal to the mid-ventral line. But this simple arrangement is modified by the development of the arm. Passing from above downwards, the modification produced by the coming limb appears as high as the third cervical (sterno-mastoid) area. On the other hand amongst the areas of the trunk the arm makes its influence felt as low as the fifth thoracic (subscapulo-axillary). Thus at least ten areas are altered from the primitive band-like arrangement by the formation of the limb.

Thus although I have chosen the sterno-mastoid (cerv. 3) area as my type it must be borne in mind that this area already bears the impress of the coming limb. Fig. 30 shows this area reduced to a rough diagram. It may be described as having a dorsal limb, a ventral limb and a third limb, the lateral flap. The angle formed by the junction of the dorsal with the ventral limb may be called the cephalic[1] angle, and in the third cervical area is practically equal to two right angles. The lateral flap lies opposite the cephalic angle and is the first expression of the changes produced by the arm.

In the sterno-nuchal area the ventral limb is somewhat attenuated. The dorsal limb is still strongly developed and the lateral flap has just reached the arm in the neighbourhood of the bicipital groove.

The fifth cervical has lost its attachment to the chest, and the ventral limb is represented by a portion on the ulnar aspect of the upper arm. The summit or extreme ventral part of this limb is represented by a spot over the upper part of the anterior axillary fold. The dorsal limb is still of fair size, but is practically detached from the lateral flap. This lateral flap is much elongated and reaches the bend of the elbow. As the dorsal and ventral limbs approximate, the cephalic angle becomes more acute. Thus in the sterno-mastoid (third cervical) area it is practically two right angles, whilst in the fifth cervical it has become an acute angle.

---

[1] I choose this term in place of the more usual "anterior" to prevent confusion. For anterior to the comparative anatomist means nearer the head, to the human anatomist nearer the ventral surface of the body.

With each successive area the anterior limb becomes smaller than the one above it, and in the sixth cervical area

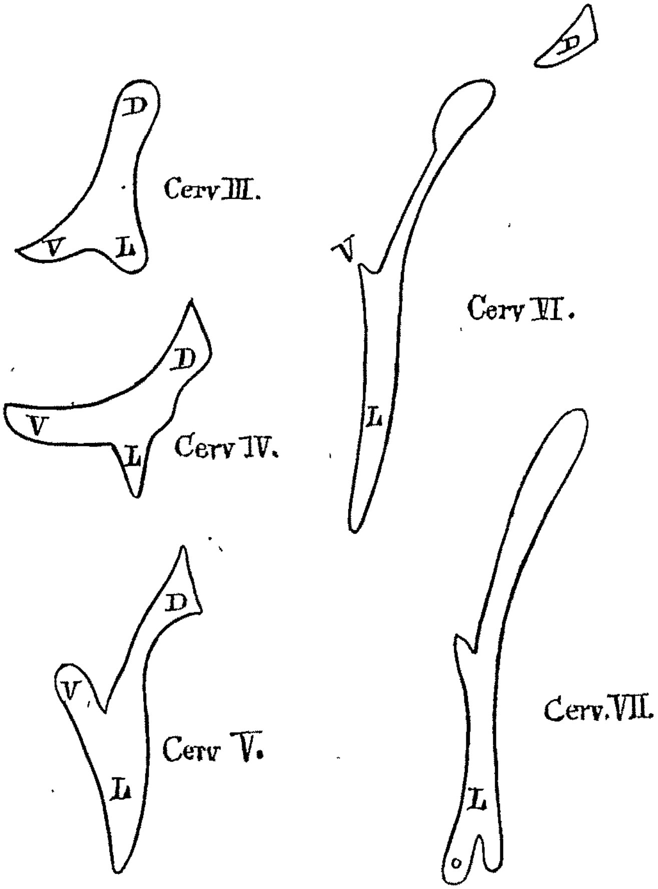


FIG. 30.

A series of diagrams to show the homologous parts of the areas from the third to the seventh cervical.

In each case D represents the dorsal, V the ventral portion of the primitive band-like area extending from dorsum to venter. L represents the lateral flap.

The seventh cervical area supplies the thumb and the posterior aspect of the index finger. The skin forming this area is therefore supposed to be stripped from the forearm and hand and spread out as a flat surface.

is reduced to a small tag. On the other hand the sixth cervical still has a dorsal limb of considerable size attached to the spine by a small dorsal flap. The greatest change, however, has taken place in the lateral flap, which is enlarged downwards and reaches the wrist.

The next area below, the seventh cervical, is no longer attached to the spine behind, but still has a considerable dorsal limb. The ventral limb is scarcely recognisable and the cephalic angle has almost disappeared. On the other hand, the lateral flap has become the most important part of the area and supplies the thumb and radial side of the hand.

Thus, as we pass downwards, area by area, the ventral attachment to the chest rapidly disappears (at fifth cervical). The dorsal attachment becomes separated from the dorsal limb and finally disappears (at seventh cervical).

The behaviour of the lateral flaps is very instructive. That of the third cervical (sterno-mastoid) lies on the ventral aspect of the shoulder joint. That of the fourth cervical has already reached the arm and lies on the ulnar aspect of the extreme upper part of the arm to the dorsal side of the lateral limb of the third cervical (sterno-mastoid). The tip of the lateral limb of the fifth cervical has shifted a little more towards the centre of the arm. That of the sixth cervical lies over the centre of the forearm; whilst the tip of the lateral flap of the seventh cervical occupies the thumb and a portion of the radial side of the back of the hand. Thus with the disappearance of the ventral limb and the increasing acuity of the cephalic angle, the tip of the lateral flap shifts gradually towards the radial aspect of the limb.

So far I have only attempted to homologise the sensory areas on the arm from the third to the seventh cervical. In order to understand the eighth cervical it will be necessary to begin from below and first consider the distribution of the fifth dorsal segment. As I showed in my first paper,[1] this area consists of a complete band round the body from back to front. But on the upper or headward side is a small flap rising towards the axilla. This represents the lateral flap.

---

[1] *Op. cit.*, p. 37.

Now my readers will see that this lateral flap differs from the lateral flap of the third cervical in that it lies on the headward side of the dorsal and ventral limbs of the band, whilst the lateral flap of the cervical areas was turned towards the thorax. Thus the primitive type of the thoracic areas,

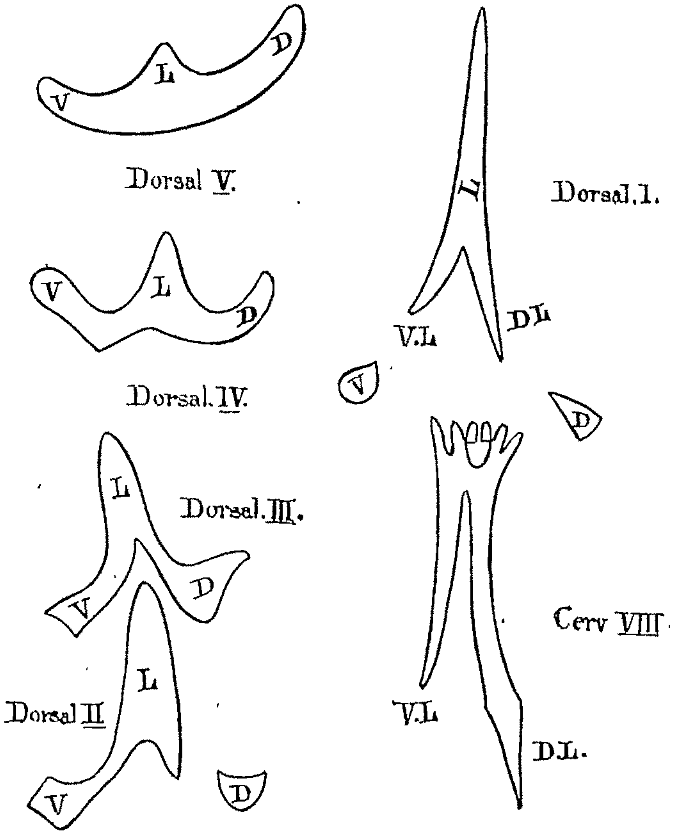


FIG. 31.

A series of diagrams to show the homologous parts of the areas from the fifth dorsal to the eighth cervical.

As before D represents the dorsal, V the ventral portion of the primitive band-like area. L represents the lateral flap. In Dorsal I. and Cerv. VIII. the angle has cleft the lateral flap, forming a ventral (V.L) and a dorsal (D.L) portion.

In each case the skin forming each area is supposed to have been stripped from the arm or hand and spread out on a flat surface. Hence that part of the hand supplied by the eighth cervical is represented like an unsewn glove. The terminal portion of the little finger thus lies in the centre without the basal phalanx which is supplied by the first dorsal area.

affected by the limb, differs from that of the cervical areas in that the lateral flap appears on the side towards the head. The position that was occupied by the cephalic angle is now occupied by the lateral flap whilst the angle is on the posterior or thoracic aspect of the band. I shall therefore speak of this angle as the thoracic angle (fig. 31).

In the fourth dorsal area the lateral flap is distinctly larger and reaches well up into the axilla, on the ventral side of the lateral flap of the fifth dorsal.

The lateral flap of the third dorsal area reaches the limb and shows a tendency to become detached from its dorsal flap whilst remaining in connection with the ventral portion of its area. Just as we saw an ever increasing acuity of the cephalic angle in the lower cervical areas, so here the thoracic angle gradually reaches a right angle.

In the second dorsal area these changes have gone still further. The lateral flap has come down the arm to the elbow, but still remains in connection with the ventral limb over the pectoralis. The dorsal limb has became completely separated as a patch over the vertebral border of the spine of the scapula.

In the first dorsal area, the lateral flap has come into the forearm, and is separated from both the ventral and dorsal portions. The remains of the thoracic angle are seen as a notch between the ventral and dorsal peaks of the lateral flap on the arm.

The eighth cervical has entirely lost its dorsal and ventral limbs, and the lateral flap is enormously elongated, and deeply cleft by the thoracic angle. It reaches the hand both back and front, and lies to the radial side of the lateral flap of the first dorsal area, both on the front and back of the forearm.

I have now traced the evolution of all the upper sensory areas from two modifications of a simple band-like type.

The areas from the third to the seventh cervical are modified by the development of a lateral flap on the thoracic side of the band, whilst the areas from the fifth dorsal to the eighth cervical arise from a type with a lateral flap on the headward side of the simple dorso-ventral band. Thus a comparison of the eighth cervical with its neighbour the seventh cervical shows that the cleft in the former represents a thoracic (posterior) angle, whilst the cleft in the latter represents a cephalic (anterior) angle.

The sensory areas on the upper limb have therefore been developed from a primitive band-like arrangement by a process that could be represented by pushing the finger from below through a sheet of india-rubber.

As Herringham and Sherrington have both pointed out the motor distribution follows a different arrangement. For the most distal segment of the limb (hand) is supplied with motor nerves which arise from the cord lower than any others entering the arm; whilst the muscles of the shoulder are supplied by those nerves which come off from the highest level of the cord.

It is interesting to notice that, according to Sherrington,[1] the highest root area which shows traces of the coming limb is the third cervical, whilst the lowest thoracic root affected by the presence of the arm is that of the fifth thoracic. In the same way I pointed out that, of my segmental areas, the third cervical (sterno-mastoid) was the first to show a lateral flap downwards, whilst the fifth thoracic was the last to show a definite lateral flap upwards.

§ 4. *To what extent do these Areas overlap?*

I shall deal with the question of overlap as a whole in the third part of this investigation. Here I shall only touch on that part of the question which is elucidated by the distribution of the areas on the head, neck and arm.

The distance between a point on the skin over the temporal crest of the frontal bone and the middle line of the forehead, at the level of the eyebrow, varies to a comparatively small extent in adult human beings. The areas on the forehead thus form a useful criterion of the amount of the overlap. If the fronto-nasal area be tender its external border reaches some three-quarters of an inch (2 cm.) from the middle line. If, on the other hand, the mid-orbital area be tender its mesial border lies about half an inch (about 1·2 cm.) from the middle line. Thus, if these two areas overlap, they do so to the extent of about a quarter of an inch (0·75 cm.). Again the posterior border of the fronto-temporal area lies about 3¼ inches (8 cm.) from the middle line, whilst the anterior or mesial border of the temporal area lies about 3 inches (7·5 cm.) from the middle of the forehead at the same level.

---

[1] *Phil. Trans.*, vol. clxxxiv., p. 750.

Now if we consider the difficulty and amount of experimental error that must attend the attempt to map out these areas clinically, a quarter of an inch (0·75 cm.) is well within the errors of observation.

Again, the posterior border of the vertical area, when well developed, extends to a point just behind a vertical line from ear to ear. The anterior border of the parietal area comes forwards just about to the same line in front.

Thus I believe that the areas of the forehead and scalp overlap to a small extent, but that the amount of overlap lies within the errors of clinical delimitation. The overlap is in no way comparable to that of the areas supplied by posterior roots. For Sherrington has shown that the areas of distribution of the posterior roots overlap to such an extent that the posterior border of A is overlapped by the anterior border of C, and B is entirely covered, in front by A, and behind by C. Thus division of one posterior root produces no anæsthesia, as was shown by Sherrington in the monkey, and by myself in man.[1] In chapter v. I shall show that the branches of the fifth nerve overlap to the same extent as the posterior roots.

The lateral areas of the cheek and jaw overlap to a slight extent, whilst the areas on the front of the neck in direct continuity with them below overlap to an extent that is clinically perfectly evident.

In the same way the third and fourth cervical do not overlap to any marked extent, whilst the areas on the arm overlap appreciably.

---

[1] *Op. cit.*, p. 41.

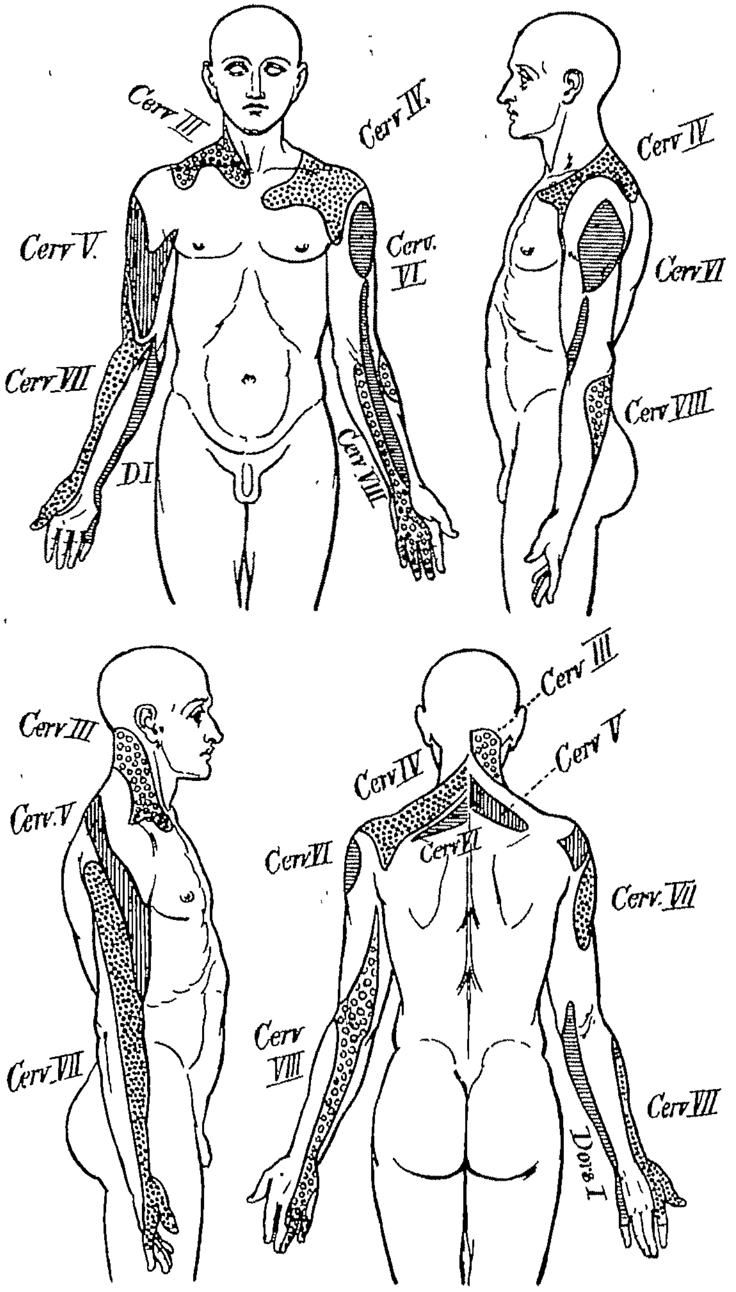


Fig. 32.

I have here attempted to show approximately the distribution of the areas from the third cervical to the first dorsal. The amount of overlap can be seen by comparing any area on the right side of the figure with that above or below it on the left side.

Thus I believe that the tender areas representing segments of the cord may overlap, but that the overlap is slight, except in those parts of the body where compression has taken place. Thus the areas on the front of the neck overlap much more than those on the side of the cheek, and the eighth cervical on the arm overlaps its neighbours above to a much greater extent than the third cervical.

For this reason the dorsal and ventral flaps of the lower cervical areas overlap to a much greater extent than the lateral flaps. Thus there is much overlapping of the crowded ventral limbs in front between the clavical and the fourth rib, and a similar overlapping of the dorsal limbs behind in the area from the fifth cervical to the second dorsal vertebral spines. These two territories might be called the "watersheds," for they form the points of separation for the streams that are flowing down the arm from above and below. This concentration explains the overlap of the dorsal and ventral limbs, so much in excess of the overlap of the lateral flap of the same area (compare the distribution of the hand from the seventh cervical, eighth cervical and first dorsal areas).

The destruction of one posterior root causes no anæsthesia, but in no case would the absence of one of these segmental areas fail to leave a gap at some point between its neighbour above and that below it in order. Thus, if even the diminished inferior laryngeal area is unaffected, a distinct gap seems to be left between the mental area which overlaps it above, and the superior laryngeal which overlaps it below.

CHAPTER II.

Pain and Tenderness produced by Lesions of Special Organs of the Head and Neck.

We have now traced the position and limits of the areas on the head and neck, which may become tender from various visceral disturbances.

In the following chapter I deal with one organ after the other in the head and neck, and discuss the locality and nature of the pain and tenderness produced by organic and functional disturbances in the activity of each organ.

§ 1. *Eye.*

Of all affections of the eye which cause pain and tenderness, some error of refraction is the commonest. A healthy young man, who has adopted some profession involving near work, begins to suffer from headache "over the eyes," or a young woman, otherwise in perfect health, finds she cannot sew without bringing on a headache. In out-patient practice the child who suffers from headache during its lessons, appears with considerable frequency in these days of Board schools.

If asked to point to the situation of the pain, these patients, as a rule, place the right hand over the forehead with the hypothenar eminence over the centre of the right eyebrow, and the second phalanges of the four fingers over the centre of the left eyebrow. Now this position of the hand has led to the common statement that the headache is "frontal." But the patient himself states that the headache is "over the eyes," and when asked to point with two hands to the position of the headache, he places the tips of the fingers over the centre of each eyebrow.

If we now pick up the skin gently between the finger and thumb the patient winces as soon as a spot is reached about three-quarters of an inch (2 cm.) from the middle line on either side. If the blunt head of a pin is used he complains that he is tender, or that he is being pricked, as soon as an area over the centre of the eyebrow is reached. By either of these methods the headache produced by hypermetropia is found to be associated with superficial tenderness over more or less of the area I have called the mid-orbital. (*Vide* p. 10, fig. 2.) As a rule in a pure case of hypermetropia the maximum only of this area is present, but occasionally, especially if the patient is seen immediately after reading or sewing, the whole area, including the patch on the front of the hairy scalp, may be tender.[1] (Case 2.)

If due to hypermetropia the headache will be found to be worse over that eye in which the error of refraction is greatest.

This headache has certain peculiar characteristics. It comes on in the morning as soon as the eyes are opened, is then most intense and gradually wears off during the day if the patient does not engage in any near work. It is intensified or produced by reading and sewing. It disappears if the eye is put under atropine.

Sometimes, when brought face to face with a headache accompanied by mid-orbital tenderness, it is not easy to say whether it be due to an error of refraction, which is undoubtedly present, or to some other cause. In such a case before correcting a slight error of refraction it is well to make sure of the following points.

(1) The headache is definitely made worse by reading and sewing.

(2) It comes on in the morning as soon as the eyes are opened.

(3) It disappears under atropine applied for a sufficient length of time to paralyse accommodation. (Compare Case 52, p. 454.)

(4) There is absolutely no pain or tenderness elsewhere in the body, and, especially, no tenderness over those areas of the body which are definitely associated with the mid-orbital. (*Vide* pp. 452 to 457.)

If, however, pain and tenderness are present elsewhere in the body, or if the patient is anæmic, it may be quite impossible to say whether the hypermetropia present is really the cause of the headache.

It is interesting to find that whilst this headache comes on in the early morning in those who lead an ordinary life, it makes its appearance in the evening in those who work at night and sleep in the day. Thus nurses on night duty who suffer from hypermetropia develop the headache on getting up in the afternoon; but as soon as they change on to day duty the headache shifts its time of appearance and comes on in the early morning.

---

[1] Patients who come to the hospital have not, as a rule, exercised the eyes with near work on the day on which they come.

Thus hypermetropia causes a well-marked headache "over the eyes," frequently accompanied by a feeling of aching "in the eyes"; and this headache is associated with superficial tenderness over more or less of the mid-orbital area.

On the other hand simple myopia, however high the error may be, causes no such headache and tenderness. The myopic sometimes complain of a "tired, aching feeling" over the forehead. This cannot be definitely localised, and is unaccompanied by superficial tenderness of the forehead. It is never present except after a long and tiring round of looking at objects at a distance (*e.g.*, picture gallery), and is always absent after a night's rest.

There is, however, one condition in which simple myopia may produce a mid-orbital headache associated with tenderness. I allude to those uncommon cases in which myopia is associated with spasm of accommodation. This headache, like that produced by hypermetropia, entirely disappears under atropine.

In progressive myopia the conditions are somewhat different. For in this disease destruction is progressing at the back of the eye. Patients suffering from this disease sometimes complain of pain "on the top of the head," and I have seen tenderness of the hairy scalp in such cases which may have been connected with the changes in the eye. Such patients far more commonly complain of a pain "at the back of the eye," especially aggravated by movement of the eye. This pain is not associated with superficial tenderness and appears in many cases to be a local, as opposed to a referred, pain.

Now, as I pointed out above, many hypermetropes complain of pain "at the back of the eye." Thus there are two separate forms of pain, both of which are described by the patient in roughly the same terms "aching at the back of the eyes." The pain associated with progressive myopia seems to be a local pain depending possibly on the condition of the back of the eye; whilst in hypermetropia the pain is referred to the maximum spot of the mid-orbital area over the upper lid, just below the centre of the eyebrow, and is therefore described by the patient as "pain in the eye." This pain of progressive myopia is not associated with tenderness over the mid-orbital area, though possibly the vertical area may be tender, whilst with hypermetropia tenderness is frequently present.

Myopic astigmatism, when uncomplicated, does not produce referred pain or tenderness. On the other hand, hypermetropic astigmatism is an even more fruitful source of headache and tenderness over the mid-orbital region than simple hypermetropia. Thus, whereas a simple error of + 1 or + 2D. can sometimes be neglected, an astigmatic error of + 1D. will frequently produce the most definite pain and tenderness.

What is the cause of this referred headache and tenderness associated with hypermetropia? The following points seem to shew that it is due in some way to the ciliary muscle. (1) Its appearance with hypermetropia and not with myopia seems to show that it is due to that part of the eye which is strained in hypermetropia, *i.e.*, the ciliary muscle. (2) Its disappearance under atropine points to the same conclusion.

At first sight the occurrence of this headache on waking in the morning would seem to tell against this hypothesis; but I think it may be explained as follows. During sleep the mechanism of accommodation comes to rest, and any spasm of accommodation, present during the day, slacks off. But as soon as the eyes are opened the tone of the ciliary muscles increases rapidly. Now the amount of pain caused by any physiological process, such as the contraction of the ciliary muscle, depends not so much on the amount of the contraction as on the suddenness with which the contraction takes place. Thus a considerable physiological effect can be produced without pain, provided the end be attained slowly, whilst the attainment of a smaller effect may act as a more violent stimulus if the height of contraction be quickly established. When the eyes are opened in the morning the curve of contraction rises rapidly and pain is produced. After a while adaptation takes place, and, although the same contraction may be maintained, the pain lessens. It is then revived by some additional strain, such as that involved in near work.

Anything which tends to produce a permanent abnormal tone of the ciliary muscle will produce referred pain. Thus unsuitable glasses may cause more headache and tenderness than the original error of refraction.

This view is supported by the absence of referred pain and tenderness when presbyopia is fully established. A patient may have been hypermetropic and have suffered from pain over the eyes all his life without appreciating that he had an error of refraction. When the error manifests itself with the onset of presbyopia, his vision fails for near objects, but his headache improves. Thus headache may be present at 40 but not at 50.

The same amount of effort which is painless with a strong ciliary muscle may become painful if the muscle is weakened, either directly or by a deterioration of the general health. Thus, headache may persist, and be associated with tenderness up till the time that presbyopia is fully established. Again, children will bear an error of + 3D. without pain if in good health. But many London children now come to our hospitals complaining of mid-orbital headache, associated with definite tenderness, for which no cause can be found beyond an error of + 1·5 to + 2D. Such children occasionally lose the headache and tenderness under a course of treatment directed towards the improvement of the general health. Many men living the tiring and insanitary life of a resident in a general hospital will develop a hypermetropic headache for the first time in spite of the fact that there is no obvious increase in the amount of near work.

Affections of the conjunctiva or of the outer layer of the cornea produce no referred pain. Injury to the surface epithelium of the cornea produces an intense smarting pain, which is well defined and local. It is exactly equivalent to the well-known local pain produced by injuries to the conjunctiva.

If, however, the deeper parts of the cornea be affected, as in deep and ragged ulcers of the cornea, or if the depth of the anterior chamber be increased, marked reference may take place. The patient complains of pain over the forehead, and more or less of the fronto-nasal area may be tender (fig. 1, p. 346). As a rule, however, the maximum only of this area is present, for if the affection of the cornea is sufficiently grave to produce superficial tenderness over the whole fronto-nasal area, some part of the mid-orbital area will also appear.

The figure to illustrate this point (fig. 1, p. 346) is taken from a case (No. 1) in which the so-called "serous iritis" formed the beginning of a fresh attack of interstitial keratitis. Now although pathologically, the ciliary body is probably at fault in the production of keratitis punctata, from the point of view of pain, I believe the deepening of the anterior chamber is the most important factor. For, as I have all along pointed out, alterations in pressure are the most effectual stimuli for the production of referred pain (associated with tenderness). Now, in keratitis punctata, opacities do not form in the vitreous as in, what is known as a true, cyclitis; but on the other hand the anterior chamber is deepened. In correspondence, I think, with this difference true cyclitis produces pain and tenderness in the mid-orbital and fronto-temporal regions, and probably even posteriorly to the fronto-temporal, whilst keratitis punctata produces pain and tenderness over the fronto-nasal and mid-orbital areas.

Case 28.—Charles S. (U.C.H., Mr. Tweedy), aged 13.

Four years ago his eyes became blood-shot. He gradually went blind and remained so for several months; he then improved, but the left eye has recently become worse.

Admitted November 21, 1892.

Not cachetic. Bridge of nose good. No scars about angle of mouth. Teeth not notched.

Lids and puncta normal.

Anterior and outer part of the globe of both eyes is of a purple colour. This is seen to be due to an inflammation and congestion of the deep structures in the region of the ciliary body. This inflammatory condition in the ciliary region is associated with the enlargement of some of the conjunctival vessels. The conjunctival, as opposed to the ciliary injection, is, however, very slight. The cyclitis is exceedingly chronic in the right eye, but somewhat more acute in the left eye.

Right cornea clear, but there is a hazy white nebula almost exactly in the centre of the left pupil.

Pupils medium size—$4\frac{1}{2}$ to $5\frac{1}{2}$ mm. in diameter; equal. Iris a fair colour. No synechiæ. Pupils react to light and accommodation well and regularly.

Vision R. eye $\frac{6}{12}$, well. Jæger 1.
L. eye $\frac{6}{60}$ (the letter is indistinct from haze), Jæger 16 (sideways).

He complained of pain over the centre of the left eyebrow and "in the left temple." This pain was associated with some superficial tenderness over the mid-orbital (maximum only) and fronto-temporal areas. This pain and tenderness was never very severe and rapidly passed off under treatment.

Thoracic and abdominal viscera healthy. Bowels open daily. No cause for pain found in any other part of the body.

Should the substance of the iris be inflamed the pain is usually said to be "on the temple" or " in the cheek." Corresponding to this pain superficial tenderness may be present over the fronto-temporal, maxillary, and temporal areas. This implication of one, or both of the two temporal areas explains, I believe, the relief caused by the application of the leech to the "temple." For the maximum of the fronto-temporal area lies just above the outer extremity of the eyebrow, that of the temporal area a little behind it; and it is close to one of these points that the leech is usually applied.

I give two cases in illustration of the tenderness produced by affections of the iris. In the first (Case 29) a foreign body was removed somewhat clumsily, and slight iritis followed in a few days. This patient suffered from pain in the left "temple" and "cheek" associated with tenderness over the fronto-temporal and maxillary areas. The second case (Case 30) was one in which iridectomy was performed for blocked pupil, and the iris somewhat extensively lacerated owing to the difficulty of the operation. In this case the patient complained of pain over the left cheek after the operation, and some tenderness was present occupying the maxillary area.

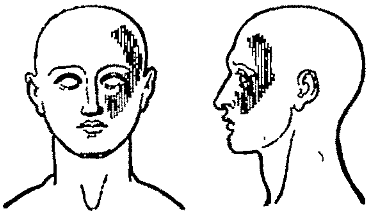


FIG. 33.

Shows the distribution of the tenderness in a case of slight iritis (Case No. 29). The areas affected are the fronto-temporal and maxillary.

Case 29.—Henry D. (Mr. Tweedy), aged 30.

November 17.—Something fell from the ceiling into his left eye whilst he was engaged on his work as a paper-hanger. He came to the casualty department and "a foreign body" was removed.

November 19.—He began to suffer from more pain.

November 21.—Ciliary injection and distinct signs of iritis. Pupil bound down. Under atropine, the pupil dilated, leaving a ring of uvea on the anterior capsule.

He complained of pain in the left "temple." Definite superficial tenderness of the fronto-temporal and maxillary areas.

The whole of the pain and tenderness had disappeared when seen a week afterwards. The left eye was deeply under atropine.

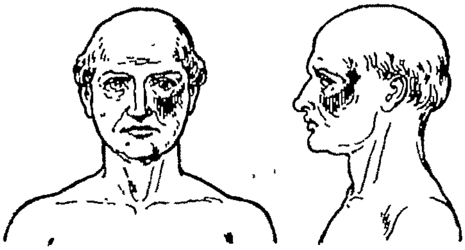


FIG. 34.

To show the superficial tenderness which followed an operation on the iris (Case No. 30). The area affected is the maxillary.

Case 30.—Thomas V. (U.C.H., Mr. Tweedy), aged 70.

About Christmas, 1891, his eyes became red and inflamed. Much headache over forehead and top of the head. In two or three weeks "inflammation" had subsided, and vision was reduced to present condition.

October, 1892, condition as follows:—

Vision, right and left, hand movement and light and darkness only; cannot count fingers.

Complete posterior synechiæ and occlusion of pupils.

Tension about normal in both eyes.

No pain; no tenderness. Pulse 80, regular; arterial wall much degenerated and very hard.

No abnormal physical signs in chest beyond those of emphysema.

Urine 1020 sp. gr., acid, no albumen or sugar.

October 6.—Iridectomy upwards in right eye.

October 20.—Good recovery and absence of pain or tenderness. An iridectomy was performed to-day on the left eye. The iris tore and the operation was more difficult and less satisfactory than that on the right eye.

October 21.—He is complaining of a little pain and there is distinct tenderness over the maxillary area (fig. 34) to the head of a pin. This tenderness was present for nearly forty-eight hours.

November 24.—Right eye, good coloboma upwards. Total posterior synechiæ. Sphincter portion of iris at coloboma still adherent. Lens seems clear, but disc and fundus cannot be seen. Vision: sees fingers vaguely, but cannot count them.

Left eye, jagged coloboma down and in. In this eye uvea only is left opposite coloboma. Lymph in pupillary area. Vision: hand movement only.

Should the tension in the vitreous chamber rise, as in glaucoma, the chief pain shifts further back, and is mainly referred to the temple. Thus in true glaucoma the pain may lie over the fronto-temporal, temporal, and maxillary areas, but the maximum pain is referred into the temporal area. All these areas may be more or less tender. The teeth of the upper jaw ache on the side affected. If the pain and tenderness spread forwards to affect the frontal area the teeth of the upper jaw ache up to the middle line, and if the pain and tenderness spread back behind the temporal area, as it not uncommonly does in an acute attack of glaucoma, the teeth of the lower jaw may also ache. Occasionally the teeth affected are tender to percussion as though periodontitis were present. That this tenderness is reflex, and not due to any actual change in the sockets of the teeth is shown by the daily variation, during the progress of a particular case, in the position and number of the tender teeth—a variation which seems to be related to the number and position of the areas which are tender on the surface of the head.

The case I have selected to illustrate these points (Case 4) was one of acute glaucoma (fig. 4). It shows this temporal area in a striking manner. Usually, however, the pain and tenderness in acute glaucoma are more widely spread, but I selected this case more with a view to illustrating the temporal area than of showing the commonest sensory changes in acute glaucoma. But whatever other areas may be affected this temporal area is the first to come and the last to disappear when the tension rises in the vitreous chamber.

Now vomiting is an exceedingly common occurrence in acute glaucoma, and I cannot help thinking that this proneness to nausea and vomiting is due to the primary implication of this temporal area. For, as I pointed out above (p. 13), and as I shall show later on (p. 107), this area is peculiarly associated with the act of vomiting, and its attendant nausea. Thus, I think it is probable that the early appearance of vomiting in acute glaucoma may be due to the fact that a rise of tension in the vitreous chamber of the eye causes tenderness over the temporal area—an area which is from other causes peculiarly associated in the nervous system with the act of vomiting and the feeling of nausea.

It is exceedingly difficult to determine what special areas, if any, are associated with disturbances of the retina and optic nerve. Detachment of the retina, if it occurs suddenly is sometimes associated with tenderness over the vertical area, and pain " over the top of the head " (Case 31). After excision of the eyeball, pain is, as a rule, absent, but such cases may sometimes complain of pain on the "top of the head," and occasionally superficial tenderness may be present over the vertical area of the scalp (Case 5, p. 15).

Case 31.—William H. (U.C.H., Mr. Tweedy), aged 19, butcher.

September 9.—His left eye "dazzled," "the place seemed foggy." A light shade appeared over the sight, which rapidly became darker. No history could be obtained of injury or of previous defect of vision in this eye.

September 11.—Came to Eye Department, U.C.H. Marked detachment of retina of left eye. Taken into hospital and put strictly to bed with eyes bandaged.

He complained of pain "which kept bobbing up from the eye to a place on the top of the head." He says the top of his head is sore and that the hair is tender.

Very definite superficial tenderness over what appears to be the vertical area of the scalp. The posterior border touches the vertical line from ear to ear, and the anterior border does not involve the extreme front of the hairy scalp. It is strictly limited to the left side of the head.

October 17.—Bandages removed. Condition of the left eye as follows: Pupil dilated. Small hazy patch at lower and outer periphery of lens. Fine dust-like opacities in lens and vitreous. Extensive detachment of lower part of retina. Retina stippled with proliferated pigment. It is pink over the greater part, but with a few grey bands here and there. Summit of the detachment seen with + 8D. Marked diminution of the lower quadrant of the retinal perimetric field of the left eye (upper projected field).

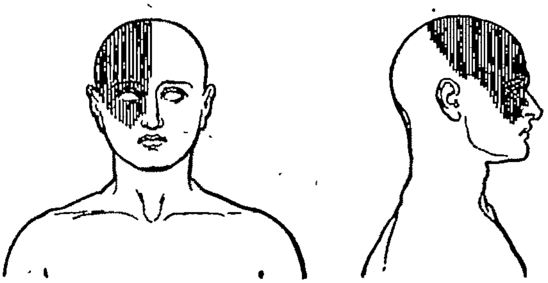


FIG. 35.

To show the extent of the skin of the scalp and face which may become tender as the direct result of lesions of the eye.

To sum up the conclusions to which we have arrived in this section.

(1) The pain associated with lesions of the conjunctiva and anterior layer of the cornea is local and not referred. The patient is guided to the position of the lesion by the situation of the pain, just as we are guided to the position of an injury to the skin by the situation of the pain it causes.

This also applies to lesions of the socket which do not involve the eyeball directly (*e.g.*, orbital growths, &c.).

(2) Lesions or disturbances in the eye from the substantia propria of the cornea back to the retina produce referred pain—that is, pain which is not felt directly at the seat of the disease—in addition, it may be, to local pain. This pain, if sufficiently acute and of some duration, is

frequently accompanied by superficial tenderness of the coverings of the head and face.

The areas that may become tender from diseases of the eye are shown in fig. 35 and extend from the fronto-nasal back to the vertical.

(3) The more anterior the lesion in the eye the more the frontal areas tend to be implicated. The further back the lesion, the more readily does the vertical area tend to make its appearance.

Deepening of the anterior chamber is associated with tenderness over the fronto-nasal area and its immediate neighbours; rise of tension in the vitreous chamber is associated primarily with the temporal area and then with those around it.

§ 2.—*Teeth.*

For the purposes of this investigation the pain and tenderness produced by disease of the teeth form a most instructive field of research. For even the most healthy suffer from time to time with toothache, and thus we are able to watch the effect produced by visceral disturbance in a person otherwise in perfect health. The second great advantage of working with the material provided by diseases of the teeth is the certainty with which we can determine the lesion that causes the mischief. The tooth is removed, split and examined, and we can note what conditions favour the production of local pain or of referred pain with its associated superficial tenderness.

Teeth stand in a curious position compared with other organs of the body, in that they lie in a cavity where the necessary conditions for the development of powers of localisation are fulfilled. It is for this reason, I believe, that a local pain will persist in the teeth later than in any other organ of the body.

The first stage of caries of a tooth consists of removal of the enamel and excavation of the underlying dentine. During this process pain may or may not be present; but provided the dentine is affected, pain can mostly be elicited by stimulation. The most effectual stimulus in these conditions is heat and cold, for in many cases where a carious portion may be cut away without pain, a jet of hot or cold water or the blast of the chip syringe will produce a twinge of pain. Now as long as the pulp cavity is not exposed, the pain remains local. The patient will complain of an aching tooth and will point to the one affected. The pain may "dart" and "shoot," but the darting and shooting are practically confined to the aching tooth. Local stimulation produces local pain. "Neuralgia" is absent and no tenderness appears in the skin of the face.

In this stage the pain produced by the tooth is exactly analogous to that produced by injuries to the conjunctiva or outer layer of the cornea—it is purely local and points to the seat of injury.

If, however, the pulp cavity is exposed, the pain alters in character and distribution. It starts in the affected tooth and darts and shoots into the face, forehead, neck or ear. Each stab of pain lasts a few moments, only to be followed more or less rapidly by a second twinge. This is the well-known "neuralgia" caused by affections of the teeth.

On testing the face or neck during this "neuralgia" some one or more of the areas I have described will be more or less tender. On the face this tenderness is superficial, but as I pointed out above, parts of the jaw, mouth or tongue may be tender at a distance from the affected tooth, owing to their intimate connection with the distribution of these areas. Thus teeth at a distance from that affected may ache and be tender to the touch.

In testing this tenderness on the forehead or upper part of the cheek the blunt head of a pin may be used owing to the presence of a firm bony substructure. But on the lower part of the cheek or neck it is better to gently pinch the skin between the thumb and fore-finger. For in these parts the head of the pin is liable to press on tender under- ying structures, and thus lead to error. (*Vide* p. 5 and 6.)

Many patients are aware of the presence of this tender- and will tell you that they first have the darting and g, and then it "seems to leave a soreness." When the tender area is reached in testing the face it is not uncommon for the patient to say "When you touch me there, it seems to make my tooth worse." They complain more often that they are "sore, tender, bruised" when touched with the blunt head of a pin than that they are being "pricked."

This tenderness is not developed until the "neuralgia" has lasted several hours, and usually disappears within 24 hours of the removal of the tooth. Under certain circumstances the tenderness may persist and spread widely, extending even on to the arm. Such cases will be dealt with in Part iii. of this investigation when I speak of generalised tenderness or "spread."

It may happen that when the patient comes under observation the pain has ceased for many hours. In such cases tenderness will probably be absent. And yet from the account given by the patient of the nature and course of the pain it was obviously referred and not local. For the purposes of scientific investigation such cases are useless. But for diagnostic purposes, the patient's account only, may frequently be of value. For referred pain is localised by the patient in the maximum spot or spots of the tender area affected. Thus given a knowledge of the distribution of the tender areas and their maxima, it is easy to determine to what area the referred pain must have belonged, and thus indirectly to arrive at the peccant tooth. For instance, pain in the ear is associated with the hyoid area, and thus if the patient tells us that he has had earache we know that he has suffered from that referred pain which is frequently associated with tenderness over this area. Now tenderness over the hyoid area is associated, as I shall show below, with disease of the second bicuspid, and first and second molars of the lower jaw. And thus we can argue back that our patient with referred pain in the ear is suffering from disease of one of these teeth.

After caries has reached the pulp cavity many differen changes can occur. The pulp and nerve become inflame and reddened, and die rapidly and steadily. But sometir this death takes place much more slowly. One part c whole of the pulp in the chamber may die, and wit nerve in one or more roots, and yet the nerve in one root may remain alive and potent for harm for a long while. Or the inflammation and death of the pulp may go on slowly under a cap of sodden dentine. If the pulp becomes fibrous or calcareous, it no longer causes referred pain. But such cases are, in my experience, frequently deceptive; for although the nerve in one root may pull out like a cotton thread, it is not uncommon to find that another root contains a small vestige of living pulp. Irregular calcification with the formation of pulp stones frequently leaves some living pulp, and is a frequent cause of referred pain. The condition known as "polypus of the pulp" is frequently associated with a living nerve. I have an impression that where the polypoid formation is large the nerve is likely to be dead; where, on the other hand, the fungating portion is small, the nerve will probably be alive. The point of the probe will settle this at once. For should the nerve be alive the least touch with the point on the protruding granulation tissue will cause exquisite pain. These cases show beautifully marked superficial tenderness of the face.

When the pulp and nerve are dead the tooth may still be a source of pain, owing to inflammation of the periodontal membrane or abscess about the tooth. But this pain, in the vast majority of instances is local and not referred,[1] and neither follows the lines indicated in this paper, nor is accompanied by superficial tenderness. When periodontitis is present the tooth frequently seems as if it were "too long," or "longer than the teeth around." Intermittent pressure (as in chewing) on such a tooth causes pain, but steady pressure relieves the pain for a while. Sometimes pressure on a tooth in this condition is said to cause "darting" pain which travels beyond the limits of the tooth. Thus, on pressing a lower bicuspid which seemed to the patient to be longer than its neighbours, the pain was said to "run along the jaw." In the same way pressure on an upper canine produced pain darting up in the line of the socket. Neither of these cases, however, referred the pain to any maximum on the face, and there was no superficial tenderness. Both teeth reacted to tapping, but not to the point of the probe, and after extraction the pulp was found to be dead and the periodontal membrane much inflamed. Possibly this darting pain may be caused by pressure transmitted from the swollen root structures to some fine nerve trunk running in their neighbourhood. For this darting differs from that of referred pain in the fact that it only occurs on direct irritation of the tooth and is not spontaneous.

---

[1] Occasionally apparent exceptions crop up. I hope to be able to deal with these shortly in a more special paper on pain due to diseases of the teeth.

Thus, in tracing dissolution of a tooth we first find local pain and tenderness produced by early caries with a healthy pulp; then referred pain accompanied by more or less superficial tenderness, with chronic inflammation and destruction of the pulp tissue; lastly, after death of the pulp, local pain and local tenderness associated with periodontitis or abscess at the root of the tooth. If this be true we are enabled to say whether pain following the filling of a tooth is due to the progress of destructive processes in the nerve of the tooth, or to periodontal irritation. For if the tooth has been carelessly stopped without sufficiently cleansing the cavity, destruction of the pulp will continue, and the pain will follow lines of reference, and superficial tenderness may be present on the face. If, on the other hand, the filling is perfect, but the periodontal membrane at fault, the pain and tenderness will be local and not referred.

I now pass on to consider the position of the pain and tenderness produced by disease of each separate tooth.

Disease of the two upper incisors causes pain, shooting up from the teeth to the forehead. This pain is associated with tenderness over the fronto-nasal area. The nose seems to swell on the side affected, and the patient complains of frontal headache or frontal neuralgia (fig. 36).

It is interesting to notice that the tenderness caused by affections of the substance of the cornea also falls over this area. Here we again meet the well-known association of the cornea with the incisors, expressed in the arrangement of the nervous system.

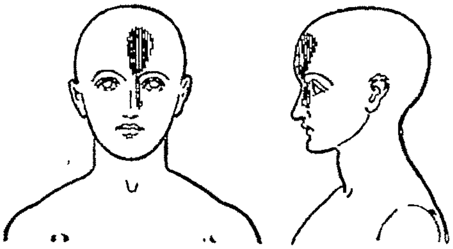


FIG. 36.

To show the area of tenderness produced by caries of the upper incisor teeth (Case No. 30). The area affected is the fronto-nasal (*cf.* fig. 1).

Case 30.—Eliza T. (National Dental Hospital), aged 34. Married.

For two years on and off the left central incisor has given trouble.

The least cold touching the tooth starts the pain. It darts up the left side of the nose to the forehead. Left side of the nose seems to swell.

When shooting pain is finished dull aching pain comes on in forehead. This is a regular dull aching headache over the front of the forehead to the left side.

Marked superficial tenderness as in fig. 36.

The left central incisor reacts to the point.

After extraction the pulp was found to be just exposed, pink, inflamed and living.

She has no pain elsewhere in body. No pain after food. No pain or tenderness of the shoulders.

Menstruation regular and painless. She is not menstruating now. Not pregnant.

The canine and first bicuspid of the upper jaw refer into the naso-labial area. Fig. 13, p. 28 shows the tenderness produced by disease of the canine of the upper jaw.

The second bicuspid in the upper jaw is somewhat anomalous. Sometimes it refers into the maxillary area, but in other cases it refers into the temporal area (fig. 37). I have never seen disease of the second bicuspid alone cause tenderness over both temporal and maxillary areas, but occasionally it distinctly affects the temporal area, and at other times equally definitely the maxillary area. Thus, this tooth seems to have an alternative nerve supply, so far as I have been able to see at present, and may cause tenderness over either the temporal or maxillary areas.

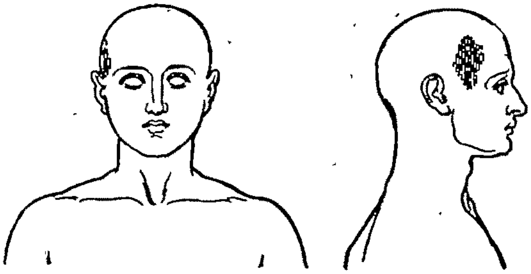


FIG. 37.

To show the area of tenderness produced by caries of the second upper bicuspid in Case No. 31. Occasionally, however, this tooth causes tenderness over the maxillary area.

Case 31.—Thomas B. (National Dental Hospital), aged 18.

He complains of pain "flying up from the tooth in the upper jaw to the right temple." He points to the second bicuspid. He says the pain seems to shoot up from the tooth deep in the jaw and then come out on to his face at the temple. He has had this pain on and off for a month. When pain is very bad he occasionally feels sick. It "turns him off his food," but he has not vomited.

Tenderness over the area in fig. 37.

No pain or tenderness elsewhere in the body. No pain at epigastrium. No cough. No wasting.

The right upper bicuspid reacts to the probe. The left lower first molar also reacts.

After extraction the pulp of the second right upper bicuspid was found to be much inflamed. Exposed, but living. No periodontitis.

Case 32.—Samuel H. (National Dental Hospital), aged 19.

June 22.—For some time he has been suffering from neuralgia over both cheeks, and much pain in the upper jaw on both sides. He went to a dentist who attempted to remove a tooth on the right side, and another tooth on the left side of the upper jaw. Since then the "neuralgia" has been much more troublesome.

Beautifully marked superficial tenderness over the maxillary area on both cheeks.

In the position of the second bicuspid on both the right and left side of the upper jaw is a stump which is exquisitely tender to the point of the probe. On the right side of the upper jaw a second stump also reacts, probably that of the first molar.

On June 22 the stump of the second bicuspid on the left side of the upper jaw was removed with some difficulty. It appeared to have been fractured. The pulp was living, reddened, and swollen, with a distinct tendency to the formation of a polyp of the pulp.

June 24.—The neuralgia has entirely disappeared from the left side of the face, but is as bad as ever on the right side.

No tenderness of left cheek, but definite superficial tenderness of right cheek, over the maxillary area as before.

The stump of the right upper second bicuspid reacts to the point as before. A similar stump belonging to the first molar also reacts to the point.

Both these stumps were removed. Both teeth have been fractured, and the pulp is reddened living and exposed. No periodontitis.

June 26.—All "neuralgia" and tenderness of the face has disappeared.

Disease of the first upper molar usually causes pain and tenderness over the maxillary area on the cheek (fig. 8).

Case 33.—Annie W. (National Dental Hospital), aged 18.

She complains of pain in the first molar on the left side of the upper jaw.

She has also had pains "in the face" off and on for a month. It has been continuous for the last five days. When asked to indicate the position of this pain in the face she places the fingers over a spot on her cheek in a vertical line dropped through the centre of the pupil a little above the horizontal level of the upper part of the ala of the nose.

Definite superficial tenderness over the maxillary area on the left cheek.

Very healthy-looking girl. No pain elsewhere in the body or head.

The first upper molar on the left side reacts to the point of the probe. No other teeth react.

After extraction both external and internal roots contained a reddened living nerve.

The second molar of the upper jaw causes tenderness over the mandibular area of the cheek. The pain is said to be just in front of the ear, slightly above the articulation of the jaw (Case 9, fig. 9).

The upper wisdom tooth (third molar) causes pain and tenderness as a rule in the mandibular area. Thus this area is shared by the two last molars of the upper jaw. Occasionally, however, the upper wisdom tooth seems to cause tenderness not only over the mandibular but also over the hyoid area.

The lower incisors are difficult teeth to investigate owing to the comparative rarity with which they are affected. Moreover, they are so rarely implicated alone that an additional element of doubt enters into the determination of their area of reference. I have, however, in the course of the last year, collected several pure cases, and believe they refer into the mental area of the lower jaw. This area is also shared by the canine (fig. 14, p. 367) and the first bicuspid of the lower jaw (case 14, p. 367).

I am still uncertain with regard to the area of the second lower bicuspid. It sometimes seems to refer into the hyoid area and sometimes into the mental area.

The first and second molars of the lower jaw cause pain in the ear and just behind the angle of the jaw, associated with tenderness over the hyoid area. (fig. 10). When this area is affected the edge of the tongue is tender to the touch on the same side as the diseased tooth (case 10, p. 361).

The lower wisdom tooth is exceedingly interesting, in that it refers over the superior laryngeal area of the neck (fig. 11). The patient complains of pain "in the throat" or neck, and not uncommonly complains that the collar rubs against a sore place in the neck (case 11, p. 363).

During eruption of the lower wisdom tooth the pain and tenderness may lie in the hyoid and not in the superior laryngeal area. This I believe to be due to the irritation of the root of the second molar by the pressure of the erupting wisdom tooth.

The widespread pain and tenderness extending down the arm to the thumb, which occasionally appears during irritation of the lower wisdom teeth, will be dealt with in Part iii., when I speak of the laws which govern the "spread" of referred pain and tenderness.

Thus to sum up the results to which we have arrived in this section :—

(1) The incisors of the upper jaw refer on to the fronto-nasal area of the forehead (fig. 36, p. 411).

(2) The canine and first bicuspid of the upper jaw refer on to the naso-labial area (fig. 13, p. 366).

(3) The second upper bicuspid refers on to the temporal or on to the maxillary area (fig. 37, p. 412).

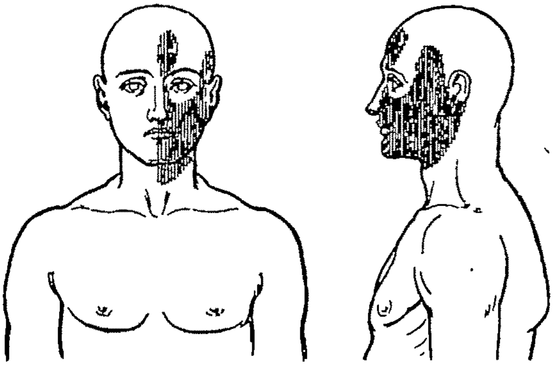


FIG. 38.

To show the extent of skin on the head and neck that may become tender from disease of the teeth.

(4) The first upper molar refers on to the maxillary area (fig. 8, p. 358).

(5) The second molar and wisdom tooth of the upper jaw refer on to the mandibular area (fig. 9, p. 359).

(6) The incisors, the canine, and the first bicuspid of the lower jaw refer on to the mental area (fig. 14, p. 367).

(7) The second lower bicuspid is doubtful, referring apparently on to the mental or hyoid area.

(8) The first and second lower molars refer on to the hyoid area and cause pain in the ear (fig. 10, p. 361).

(9) The lower wisdom teeth refer on to the superior laryngeal area of the neck (fig. 11, p. 363).

§ 3. *Ear*.

The pain produced by lesions of the auditory meatus, from its opening to the membrana tympani, is local, although frequently severe.

If, however, the membrana tympani or middle ear be affected the pain will be referred to some point at a distance from the source of irritation and may be accompanied by superficial tenderness.

When the membrana tympani is affected the middle ear generally diseased. But in some cases where an accumulation of cerumen in the meatus is decomposing, the membrane is found to be reddened and inflamed. In many cases the redness will subside after removal of the plug, and there is no reason to suppose that the middle ear is affected. Some cases of this kind suffer from pain "in the ear and under the jaw," and occasionally superficial tenderness is present over the hyoid area.

During suppuration in the middle ear before perforation of the membrana tympani occurs, the pain is intense. In hospital practice patients mostly wait until the membrana tympani is perforated, and the pain has somewhat abated, before finding their way to the ear department. Thus it is extremely rare to see an adult in this condition. The history given by such patients points to the presence of pain "at the top of the head" and "in and behind the ear." The pain on the top of the head abates when the discharge appears, but the pain "behind the ear" remains. Occasionally, however, the orifice, once established, becomes blocked. If this should occur the patient begins to suffer from pain on the top of the head, and in such cases I have seen superficial tenderness over the vertical and parietal areas of the scalp.

Case 34.—Emma R. (U.C.H., Mr. Pollard).

She has been slightly deaf on and off for the last five to six years, ever since typhoid fever. She has had a slight discharge from the left ear which ceased about June 2.

June 4 and 5 the left ear was very painful and discharged a little.

June 8.—Pain behind the jaw, in the ear and over the top of the head. Somewhat wide-spread and not very acute tenderness over the vertical, parietal and hyoid areas. The lobule of the ear is tender and the meatus is intensely tender.

Left ear: Small perforation through which no discharge is coming. Meatus not excoriated.

Right ear: Membrana tympani destroyed. Granulating spot on posterior part of inner wall of tympanum.

She improved but relapsed again, and on June 29 there was an enlarged gland behind the ramus of the lower jaw on the left side.

When a free opening is established inflammation of the membrana tympani may persist, and this I think accounts for the frequent persistence of referred pain and tenderness over the hyoid area after an acute attack of otitis media.

Thus for practical purposes the hyoid area is the most important area in ear disease. One maximum lies in the meatus and the second just behind the angle of the jaw, and the patient complains of pain at these two spots. Now, as the pain is situated in the meatus, the referred pain and tenderness are not likely to lead the ear surgeon astray when he is dealing with a case of ear disease.

But this area is shared by the posterior teeth of the lower jaw, by the tonsils, the lateral aspects of the tongue; and disease of any of these structures may cause pain in the ear. Two cases have come under my notice in which pain in the ear, accompanied by tenderness over the hyoid area, which were not due to ear disease, led the patient to consult an aural surgeon. The first of these cases came to the ear department at University College Hospital (Case 35). Mr. Pollard pronounced the ear to be healthy and all pain and tenderness disappeared when the nerve of the carious second molar on the same side of the lower jaw was destroyed with an arsenical dressing. The second case fell into the hands of an ear specialist, who perforated the membrana tympani without letting out pus or relieving the patient. Complete relief was obtained after removal of a carious lower molar with a living inflamed pulp.

Case 35.—Joseph William C. (U.C.H., Ear Department, Mr. Pollard), aged 17.

May 25.—He complained of pain in the right ear and pain behind the angle of the lower jaw on the right side. There was "tenderness over the mastoid." Mr. Pollard could find no disease of the right ear. He was treated with fomentations over the ear and jaw for four days.

May 29.—He says the pain is as bad as ever. Mr. Pollard could find no signs of disease in the ear and kindly allowed me to see the case.

There was well marked superficial tenderness over the hyoid area (closely resembling fig. 10).

He was taken to the National Dental Hospital, where the first right lower molar was found to be in the following condition:—The pulp cavity was empty, and there was secondary dentine in the posterior canal. The upper part of the nerve in this root was mummified, but lower down the nerve was living, inflamed, and exquisitely tender. An arsenical dressing was applied.

June 1.—All pain and tenderness gone. The tooth was filled.

June 2.—Absolutely no pain or tenderness. All the so-called "mastoid tenderness" has disappeared.

To sum up the results to which we have arrived in this section—

(1) The meatus auditorius does not cause referred pain and tenderness, but the pain produced by lesions of this part of the ear is local and points directly to the seat of the disease.

(2) Lesions of the membrana tympani and middle ear cause referred pain, accompanied by tenderness over the hyoid area.

(3) Elevation of tension in the middle ear, such as must occur in the first stage of otitis media before the membrane is perforated, cause pain and superficial tenderness over the vertical and occasionally over the parietal area of the scalp, in addition to that over the hyoid area.

§ 4. *Tongue.*

It is a well-known fact in surgery that malignant disease of the tongue may cause pain in the ear or over the back of the head, in addition to the local pain in the tongue itself.

Now, as I pointed out in chap. i. (pp. 360, 362, 367) each half of the tongue can be divided, with regard to its nervous connections, into an anterior, a lateral, and a posterior portion. Each of these portions stands in connection with a different area of tenderness. Thus, the anterior portion of the tongue belongs to the mental area, the lateral part to the hyoid area. The posterior part of the tongue is too far back to test satisfactorily, but we shall see in this section that it is probably related to the superior laryngeal area.

Now, it is exceedingly difficult to obtain pathological lesions which involve one only of these areas on the tongue. For tubercular ulceration is rarely of sufficient depth to cause referred pain; malignant disease is rarely confined to one part only, and is far commoner on the edges than on the dorsum; and gummata, which tend to affect the dorsum, are decidedly rare. Thus, we are practically reduced to the pain produced by malignant disease of the tongue.

If the disease involves the anterior part of the tongue pain is complained of over a spot close to the point where the mental nerve rises through the deeper structures of the jaw to supply the skin. This, as we saw (p. 367), is the maximum spot of the mental area. This pain in the front of the lower jaw is sometimes associated with superficial tenderness over the mental area (fig. 14).

Case 36.—Frances F. (U.C.H., Mr. Heath), aged 63.

For several months she has had pain in the right side of the front of the tongue and in the front of the lower jaw. For the last six weeks this pain has been very troublesome.

February 3, 1892. Beneath the right edge of the front of the tongue, just above the ridge formed by the sublingual gland, about half-an-inch behind the orifice of Wharton's duct, is a small ulcer, surrounded by a rim of indurated tissue. The centre of the ulcer forms a deep pit, with everted edges. On the right border of the tongue above this spot some ulceration and patches of leukoplakia are seen. No enlargement of lymphatic glands.

She complains of pain over the right side of the anterior part of the lower jaw, a little behind a line drawn vertically downwards from the angle of the mouth. She also has pain in the tongue itself. Around the area of pain in the jaw there is distinct tenderness of the superficial structures both to the head of a pin, and when the skin is picked up between the fingers. This represents the mental area partially developed.

The anterior part of the right side of the tongue was removed by Mr. Heath with Paquelin's cautery.

Microscopical examination showed the growth to be a squamous epithelioma.

If the lateral part of the tongue only is implicated the patient complains of pain "in the ear," and behind the ramus of the lower jaw on the side affected. In such cases the hyoid area is not uncommonly found to be tender (fig. 39). I have no case in my collection showing complete tenderness over this area alone, and I have selected one in which the main tenderness lay over the hyoid area, but with implication of the superior laryngeal area in addition.

Case 37.—Thomas C. (U.C.H., Mr. Heath), aged 68.

About March, 1891, he became "hoarse"; then gradually a small lump appeared on the tongue like a pea.

On admission, November 7, 1891. He is thin, but not

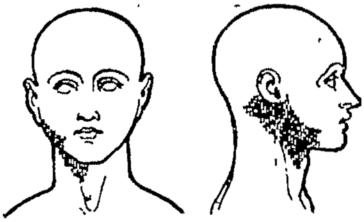


FIG. 39.

To show the area of tenderness due to malignant disease of the tongue in Case No. 37.

markedly wasted. Cannot protrude tongue to full extent. On right side of tongue about middle of its length is an ulcer which can just be filled by the tip of the finger. Exceedingly painful. Beneath the base of the jaw just opposite the situation of the ulcer, inside, is a hard secondary gland.

He complains of pain in the right ear and behind the angle of the right half of the jaw.

There is a marked superficial tenderness over the hyoid and superior laryngeal areas on the right side (fig. 39).

Mr. Heath removed about two-thirds of the right half of the tongue.

About the middle of the tongue is a shallow ulcer with raised edges. Section across the centre of this ulcer shows a yellowish white malignant mass which has invaded the tongue to the depth of at least half-an-inch (1·25 cm.). The sublingual gland is implicated by direct extension.[1]

November 27. External wound entirely healed. All pain and tenderness gone.

If the lesion is situated on the dorsum of the tongue behind a line about 3 to 4 cm. from the tip, the pain is "in the throat" close to the cornu of the hyoid bone. It may also be felt in the back of the head in addition. Occasionally tenderness is present, more commonly in the territory of the superior laryngeal, more rarely in that of the occipital area.

To illustrate the whole area that may be tender in disease of the tongue I give fig. 40. This figure shows the areas of tenderness which were present in the same case of malignant disease of the tongue on two separate occasions.

Case 38.—Richard R. (U.C.H., Mr. Barker), aged 64.

About four months before admission he began to have a little pain in the tongue. Soon afterwards he noticed a small lump, the size of a pea, on the centre of the dorsum of the tongue. This lump gradually increased in size and about a month before admission, formed a sore.

---

[1] This is a source of fallacy that I fully recognise in these cases.

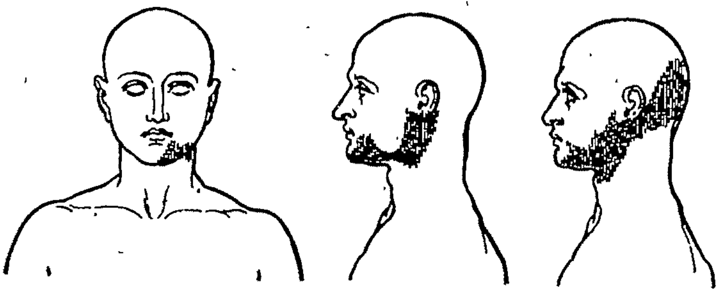


FIG. 40.

To show the areas of superficial tenderness which were present in a case of malignant disease of the left side of the tongue on two separate occasions (Case No. 88).

Three or four weeks ago he began to suffer from earache and from pain in the front of the lower jaw, just below the angle of the mouth. Sometimes the pain became very acute and is like a stab from a knife in the ear. He occasionally suffers from pain over the occipital region on the left side of the head. This he describes as a "gnawing, rheumatic pain."

All the teeth from the central incisor back to the third molar in the left half of the lower jaw ache. The second bicuspid aches most of all these teeth.

On admission (*June* 13).

Fairly nourished. No marked wasting.

On the left half of the dorsum of the tongue is a large ulcer with irregular, raised edges. The floor of the ulcer is deeply depressed and covered with a thin slough. The ulcer extends backwards from the tip of the tongue for at least 3·5 cm. The median border extends close up to the middle line of the tongue over the anterior part of the dorsum, but does not cross the middle line.

There is definite superficial tenderness over the areas shown on the two left hand drawings of fig. 40.

No teeth react to the point of a probe.

June 18.—He is still complaining of pain in the left ear, and front of jaw on left side. But he now has pain over the left occipital region, and in the region of the cornu of the hyoid bone on the left side. Swallowing causes some discomfort on the left side of the neck.

Tenderness is present to-day over the areas shown in the right hand drawing of fig. 40.

Thus at one time or another the mental, hyoid, superior laryngeal and occipital areas were tender; corresponding, I believe, to the implication of the anterior, lateral and posterior portions of the tongue.

§ 5 *Tonsil.*

When the tonsils are affected pain appears in the ear, and behind the angle of the jaw. If the disease is acute and associated with a rise of temperature, wide-spread pain and tenderness may result. This phenomena has been touched on in my previous paper (page 109), and will be treated in full when I discuss the causes of "generalisation" in Part iii.

But if there is no rise of temperature the pain produced by disease of the tonsil behaves like the referred pain from any other organ of the head. It is situated in the ear and behind the angle of the jaw on the side affected, and is often accompanied by superficial tenderness over the hyoid area. In order to avoid the complications produced by the presence of fever, I have chosen as my instance a case in which the left tonsil was in process of destruction from malignant disease (fig. 41).

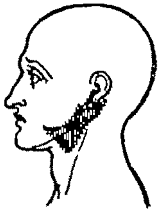


FIG. 41.

To show the area of tenderness produced by malignant disease of the left tonsil (Case No. 39).

Case 39.—Arthur G., aged 50. Married.

For some time he has had a "sore throat." The pain in the throat has gradually increased and during the last three weeks he has noticed that his beard has been sore on the left side.

Probably primary syphilis in 1875. No secondary symptoms.

He has been getting thinner for a month.

June 8, 1893. He complains of pain in the left ear and behind the left angle of the jaw.

Definite superficial tenderness over the hyoid area. The meatus auditorius is extremely tender (fig. 41).

The left tonsil is infiltrated with growth, apparently malignant, producing an ulcerated surface. No secondary glands.

§ 6. *Nose.*

But few nasal affections are painful; and even in those, that cause pain, the pain is but rarely acute.

In inflammatory conditions of the olfactory portion of the nose above the middle turbinate bones, pain associated with slight superficial tenderness appears over the fronto-nasal or mid-orbital areas of the forehead. Inflammation of that part of the nose associated with the lachrymal duct, so common in eye practice, occasionally lead to mid-orbital headache and tenderness (fig. 42, case 40).

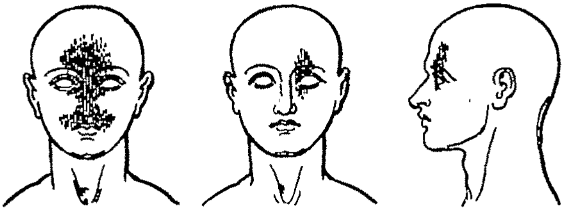


FIG. 42.

To show the tenderness which may be produced by lesions in the nose. The figure on the left hand represents the tenderness which followed an operation on the inferior turbinate bones (Case No. 41). The two figures on the right hand represent the tenderness in a case of rhinitis (Case No. 40).

Case 40.—Robert L. (U.C.H., Mr. Pollard), aged 26. Shop assistant.

August, 1891. He has been suffering from pain in the left nostril and pain over the left eye.

He cannot breathe properly through that nostril, and a whitish discharge sometimes comes from it.

Left nostril: Mucous membrane swollen and covered with glairy mucus. Septum deflected a little to the left, so that it comes in contact with the inferior turbinate. Fauces congested, pharyngeal mucous membrane swollen; one or two patches of mucus.

Ears inflated; hearing good.

August 20. Galvano-cautery to left nostril.

December 31. He still complains of pain over the left eye and there is superficial tenderness as in fig. 42.

Left nostril: Mucous membrane swollen. Excessive mucous discharge. Congestion of upper part of left nasal cavity. Turbinate bones come in contact with the septum. Plenty of room in the respiratory portion. The inferior turbinate no longer touches the septum owing to the cauterisation.

No hypermetropia.

No pain or tenderness elsewhere in the body or head.

Inflammatory conditions of the respiratory portion of the nose and of the posterior nares, occasionally, though rarely, cause pain and tenderness over the naso-labial area. If this area is tender, the patient has a feeling as if his nose and upper lip were swollen (case 41).

The connection of inflammatory conditions of the pharynx and posterior nares, with herpetic eruptions over the naso-labial area is worth notice (*vide* p. 384).

Case 41.—Alice H. (Victoria Park Chest Hospital, Dr. Thorowgood), aged 23. Single.

February, 1890.—Had "influenza" followed by "bronchitis." This lasted on and off for three months. At the end of this time she began to have attacks of true spasmodic asthma.[1]

In the course of the examination which was made after her admission to the hospital, it was found that she was unable to breathe through the right nostril, and that she breathed with great difficulty through the left nostril. The mucous membrane over the inferior turbinates was swollen.

Mr. Hovell kindly saw her at the London Hospital and found she had hypertrophic rhinitis and enlargement of the posterior extremity of each inferior turbinate bone sufficiently great to interfere with respiration.

December 2, 1893. Mr. Hovell operated after spraying the nostril with 2 per cent. cocaine. He passed his left fore-finger into the naso-pharynx and introduced a snare into the right nostril. The enlarged extremity of the turbinate body was fixed into the wire and the snare tightened. He thus removed the hypertrophied mass from the right nostril. The mass was removed from the left nostril by a similar procedure.

At two p.m. the same day she complained of a feeling as if her nose and upper lip were swollen and she had some pain over the forehead. Marked superficial tenderness was present over the areas, in fig. 42. This had entirely disappeared by the afternoon of the next day.

Thus in conclusion:—

(1) Affections of the olfactory portion of the nose may cause referred pain and superficial tenderness over the fronto-nasal and mid-orbital areas.

(2) Affections of the respiratory portion of the nose and posterior nares may occasionally cause referred pain and tenderness over the naso-labial area;

(3) But the majority of nasal affections do not cause referred pain and superficial tenderness.

---

[1] This case will be published in full in my paper on "Mental States in Visceral Disease."

§ 7. *Larynx.*

Affections of the larynx, though producing much discomfort and local pain, do not in the majority of cases produce referred pain and tenderness. The inflammatory conditions, apart from syphilis and tubercle, cause great local soreness, but as a rule the soreness is definitely in the larynx.

But where the larynx is in process of destruction by tubercle, the pain is not only local, but referred, and superficial tenderness may be present over one or both of the areas I have called the superior and inferior laryngeal. When the upper of these areas is tender, the pain is situated a little anteriorly to the cornu of the hyoid bone; when the lower area is affected the pain lies just in front of the anterior border of the sterno-mastoid muscle at about the horizontal level of the cricoid cartilage.

Thus, if we had to depend on pain only, it would be quite impossible to say whether it were local, or referred. That it is not truly local is shown by the presence of tenderness when the superficial structures of the neck are picked up between the finger and thumb. To demonstrate these areas in the neighbourhood of the larynx, the blunt head of a pin must not be used, for the pressure thus exerted will bring out deep tenderness in the structures of the larynx in addition to any superficial tenderness that may be present.

Syphilis and malignant disease are a less fruitful source of referred pain and superficial tenderness than tubercular disease. In many cases, however, that are otherwise painless, pain and superficial tenderness are produced by the manipulations necessary for treatment. Thus, in the earlier stages of tubercular infiltration when the mucous membrane is swollen, no referred pain is present. But treatment of the larynx with lactic acid will not uncommonly produce some referred pain and superficial tenderness of the skin of the neck.

If either the upper or the lower laryngeal area be tender, especially if the tenderness be acute, stimulation over the neck within the tender zone produces a feeling of tickling in the throat, and each such stimulation is frequently followed by a cough. It is to these cases that applications to the neck do so much to soothe pain and irritation produced by laryngeal disease.

The laryngeal disease which causes these areas of tenderness is usually so wide-spread that it is difficult to say whether one area belongs to one part of the larynx and the other area to another. However, I cannot help thinking that where the disease lies mainly in the region of the epiglottis and aryteno-epiglottidean folds, the upper area tends to be mainly affected (Case 42). In those cases where the cords themselves are mainly affected I think the inferior laryngeal area is more likely to be tender (Case 12).

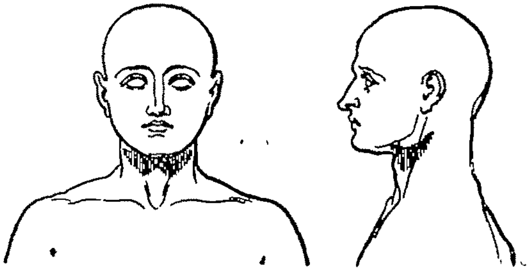


FIG. 48.

To show the areas of tenderness which were present in consequence of tubercular disease of the upper part of the larynx (Case No. 42).

Case 12, p. 364, illustrates the presence of the inferior laryngeal areas of tenderness as a result of tubercular disease of the larynx.

Case 42.—Henry H. (Victoria Park Chest Hospital, Dr. Heron), aged 26, clerk.

Quite well till the winter of 1890; then had what he calls "influenza"; pains all over, especially in the back; three weeks in bed, said to have bronchitis, never lost cough since. The next winter an exactly similar attack; sputum blood-stained. Each subsequent winter had a similar attack which lasted longer and was more severe than that before it.

Winter, 1893-94, lost his voice for the first time.

When admitted in February, 1894, there were signs of cavitation at the upper part of the right lung, with weak breath sounds, increased vocal resonance and numerous râles at right base, signs of consolidation at the left apex.

Condition of the Larynx.—Voice now fair, but he has on several occasions lost his voice. The epiglottis is greatly swollen and œdematous. The mucous membrane over the arytenoids is much swollen and pale. The cords cannot be fully seen owing to the amount of swelling. Those portions which can be seen are not ulcerated or swollen.

He has a good deal of pain in the throat, especially on swallowing.. No superficial tenderness.

He gradually developed definite superficial tenderness mainly over the superior laryngeal area, and on March 20 it was solely over this area, as shown in fig. 43. By this time marked ulceration of the epiglottis had begun, and by April 5 the right side of the epiglottis had been eaten away, and there was a ragged ulcer on both arytenoids in the region where they touch one another.

Signs in the lungs advanced down the left side. He rapidly lost weight, and could only be fed under the cocaine spray.

To sum up the conclusions to which we have arrived in this section:—

(1) The majority of affections of the larynx do not cause referred pain or superficial tenderness.

(2) Certain destructive lesions, notably tubercular disease, are associated with referred pain, and this referred pain is accompanied by superficial tenderness over one or both of the areas I have called the superior and inferior laryngeal.

§ 8. *Brain.*

The headache in cases of cerebral disease varies enormously. It is mostly wide-spread over the forehead, temples and back of the head. It may affect the cheeks, neck and shoulders. It is frequently bilateral, and thus apparently loses all localising value, not even helping us to diagnose the side of the lesion.

Ferrier first pointed out that pressure on the skull frequently caused pain in cases of cerebral tumour. Since he described this tenderness on pressure, physicians have obtained such variable and discordant results that much less importance has been attached to this observation than it deserves. This is, I believe, due to a want of discrimination between deep and superficial tenderness. Ferrier described deep tenderness, which when present is an admirable localising sign.

In a typical case of brain tumour the patient is liable to attacks of headache associated with vomiting, and to a variable amount of persistent pain between these attacks. If the skin of the head is tested either during, or directly after, such a paroxysm of pain, more or less intense superficial tenderness may be present, exactly analogous to that which I have described in diseases of other organs of the head. It is generally wide-spread and not localised to small patches. If the patient suffers from pain in the inter-paroxysmal periods, this pain is also frequently associated with superficial tenderness. Obviously, then, such pain must be referred and not local.

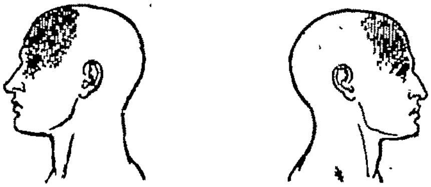


FIG. 44.

To show the superficial tenderness which accompanied the headache due to a tumour of the choroid plexus of the brain (Case No. 43).

Case 43.—Alfred C. (Queen Square, Dr. Buzzard), aged 39. Butler.

Admitted February 22. Died June 2, 1893.

About a year ago he was first noticed to be ill. He was always sleepy and tired, and suffered from bad headaches.

Summer, 1892, he began to lose his memory and wandered about as if he did not know where he wanted to go to; got into wrong trains, &c.

The attacks of headache have occurred almost daily for the last few weeks, and are frequently accompanied by vomiting.

He has never had a fit at any time.

No history of alcohol (Butler) or syphilis. Children healthy.

On admission—Stout, thickly-built man of 5ft. 6in.

Face pale, complexion muddy. No œdema, anæmia or signs of general disease.

Mental condition varies considerably from day to day. His memory was never good, but is worse on some days than on others. He is despondent; bad-tempered; difficult to manage. Quickly loses his temper and fights with the other patients. No delusions or hallucinations. No grandiose ideas.

He suffers from periodic headaches. They occur at irregular intervals and come on at all times of the day. No warning. The pain is mostly over the right side of the forehead and front of the scalp. When the headache is present, combing or brushing the hair on the right side of the front of the scalp gives him pain. He generally sits up and holds his head, pressing it or putting cold water bandages upon it. Going up and downstairs increases the headache slightly, but he can continue to carry trays up and downstairs, even when the headache is bad.

Sometimes he vomits immediately, sometimes not for three to four hours. In fact, he is sometimes obliged to make himself vomit to ease the pain. The headache leaves him as soon as he vomits. He loses all appetite during the headache, but feels very hungry when the attack is over. They nearly always come on after a few days' constipation. During the attack he is giddy and sees "tiny stars." No hemiopia or other visual phenomena, such as appear in megrim.

Speech unaltered.

Writing shaky and not so good as it used to be.

He can walk and stand. Does not stagger with his eyes closed. Can toe and heel a line on the floor. Grasp right 50, left 60. All movements of legs and arms well performed. No paralysis or weakness of any group of muscles. No wasting. No tremor.

No pain or tenderness anywhere except in the head.

Sensation to touch and pain good. No alteration in sense of position.

Knee-jerk equal, not exaggerated or diminished. No ankle clonus. Wrist-tap and elbow-jerk normal and equal.

Plantar and abdominal reflexes normal.

No ptosis. No ocular paralysis. No nystagmus. Pupils about 5 mm. in diameter; react to light and accommodation. Convergence good.

Sensation and motion in the face good.

Tongue comes out straight. Not wasted. No fibrillary twitching.

Palate moves well. Voice good.

Vision right and left $\frac{6}{12}$. Jæger 1 badly.

The disc and fundus oculi are normal. No neuritis. No hæmorrhages.

Smell good on both sides. Taste bad on the protruded tongue (not pathological), but present on the palate.

Hearing very good. Watch heard at a distance of one meter, right and left.

Spine and sphincters normal.

No abnormal physical signs in the thorax or abdomen.

Urine, sp. gr. 1030, acid. No albumen.

March 22. He has had several attacks of headache since admission, in which superficial tenderness was present.

He had an attack this morning associated with the tenderness shown in fig. 44. He vomited. Bowels constipated. Tongue moist but furred.

There is no pain or tenderness elsewhere in the body or neck.

He seemed to improve and left the hospital, but died April 2, 1893.

Post-mortem.—The ventricles were much dilated. To the right choroid plexus was attached a body the size of the tip of the little finger. It contained thick, curdy material. This cyst lay close to the foramen of Monro. The rest of the brain was healthy except for the dilatation of both lateral ventricles.

But a second class of case exists in which no such superficial tenderness accompanies the headache. The pain is localised within a fairly small area, and over this area pain is produced when deep pressure is made upon the head. This pain and tenderness is definitely local and corresponds to the local pain produced by pressure on a tooth affected with periodontitis.

Case 44.—Abraham W. (Queen Square, Dr. Hughlings Jackson), aged 35. Sweep.

At the age of 15 he had a fit as he was rolling down a bank. Began in left leg; then his head felt as if it were going round, and he lost his senses.

At the age of about 21 he had a second fit. It began in the left leg, and affected the left arm.

A year afterwards had a third fit, with loss of consciousness; eight to nine months after, a fourth fit, with less profound loss of consciousness.

In the last four years he has had several attacks daily, beginning in the left leg, but without loss of consciousness.

There is no history of injury or syphilis.

No family history of fits.

On admission, June 30, 1893. Strong, healthy-looking, muscular man. Very intelligent. No mental symptoms. Speech normal; weakness of left arm and left leg, compared with the right side. Fine movements of the left hand defective and slow. Cannot unbutton his shirt easily with the left hand. The movements of the left shoulder are a little weaker than those of the right. He has slight power of movement only in the toes of the left foot. Flexion of left ankle poor; extension somewhat better, but not good. Movement of left knee powerful, but not so strong as right. At hip, and on left side of trunk, movement seems good. There is distinct loss of sense of position and some diminution to touch over the left leg. Sensation of pain, heat and cold unaffected. Optic discs normal. No paralysis of any cranial nerve. Left knee-jerk exaggerated. Right knee-jerk normal. Ankle clonus obtained on left side only. Right plantar reflex present; left plantar brisk.

From July to September he had a large number of fits, in which he did not lose consciousness. They affected the left leg and left arm, and the leg was distinctly paralysed after a fit.

September 19. Mr. Gunn thought that optic neuritis had appeared, and Mr. Horsley determined to operate.

He has complained all along that there is a sort of darting pain in the right parietal region just before the fits. For the last few days, however, he has complained of a dull, aching pain principally over the parietal eminence. This pain remains a dull ache unless he has a fit; with each fit there is a stab of pain at this point.

There is no superficial tenderness of the scalp now or at any other time. There is, however, to-day distinct tenderness on deep pressure at a point in the parietal region 1½ inch (3·75 cm.) behind a vertical line from ear to ear, and 1 inch (2·5 cm.) from the middle line of the scalp. The spot is very localised, and is not bigger than the top of the thumb. Pain can be produced either by deep pressure or by tapping (percussion).

September 27. Mr. Horsley operated and removed a considerable portion of the bone over the parietal and vertical regions. At what appeared to correspond to the ascending parietal gyrus there was a dark purple patch of injected dura about 1 cm. in diameter, corresponding to the position of tender spot on the scalp. The dura was not opened.

October 3. At the second stage of the operation by Mr. Horsley, a deep red growth was seen in the sulcus, supposed to be the fissure of Rolando, midway between the site of the genu and the margin of the hemisphere. The growth was found to penetrate for a depth of 1¼ inch (3 cm.). It was soft and friable on the surface, but very hard in the corona radiata.

Now, as far as my experience goes at present, these two forms of tenderness in brain disease are fundamentally different in origin. The deep tenderness and the definitely ocal pain are of meningeal origin, and point either to a primary or secondary affection of the dura mater. On the ther hand, the superficial tenderness associated with referred pain is due to some affection of the brain itself.

But on examining the records of cases of tumour affecting the substance of the cerebrum or cerebellum, a certain number of cases appear in which neither deep nor superficial tenderness were present. Thus we cannot say definitely that if superficial tenderness is absent there is no gross brain disease. On the other hand, the pain and superficial tenderness produced by a tumour that is too large to be excised, will disappear if a considerable portion of the vault of the skull be removed, as is usual in modern brain surgery. Brisk purgation (with a drop of croton oil) will also ease the pain in cases that have not been trephined.

Thus the referred pain and superficial tenderness cannot depend primarily on the destruction of the brain tissue, but on some condition that can be relieved by extensive trephining or brisk purgation.

Now the only condition that can be relieved by such procedures is heightened tension. Thus I believe that referred pain and its accompanying superficial tenderness of the scalp are due, not to the implication of the brain substance directly, but to the rise of intra-cerebral tension, produced by the destructive process.

We have now reached a useful and necessary distinction between the two forms of pain and tenderness. Local pain and deep tenderness point to an affection of the dura mater, either primary or secondary. Referred pain and superficial tenderness point to a rise of tension within the brain itself.

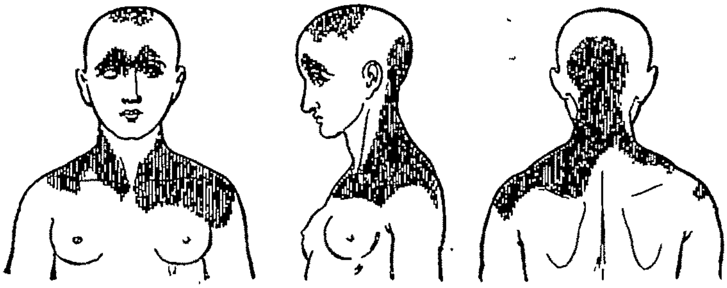


FIG. 45.

To show the areas of tenderness which were present in a case of cyst of the cerebellum (Case No. 45).

Case 45.—Sarah F. (Queen Square, Dr. Tooth), aged 21, single. Admitted August 24, 1892. Died September 18, 1892.

October, 1891. Began to have bad headaches daily. About the same time she began to vomit; headache always better after she vomited.

In about four months both headache and vomiting passed off, but her "head felt turning round," and she was giddy.

About six months ago the headache came on again, with vomiting every few days.

Three months ago the headache had become very severe, and radiated over the back of the head to the shoulders.

October, 1891. Her sight began to fail. This steadily became worse until three weeks ago, when she became blind.

She has never been ill until twelve months ago. Menstruation began at age of 16; every four weeks until the last two months, which have been missed; not painful.

A brother and a sister both have phthisis.

Condition on admission.—Small girl of 21. Dull, expressionless face, except when suffering from pain. No very marked wasting. Pulse 88. Respiration 21. Temperature 98°—99·4° F. (36·7°—37·4° C.).

Mental condition fair, except when she is suffering from an attack of pain.

She suffers from violent attacks of pain in the head, worse on the left side than the right. During the attack she lies on her right side with her head buried in the pillow, crying out with the pain. The pain is most constant in the occipital region, but is also felt over the forehead, temples and vertex, and sometimes extends down the neck to the shoulder. These attacks are associated with vomiting; she feels sick for five to ten minutes before the vomiting occurs. The pain is less severe after she has vomited.

In between the attacks the back of her head and neck show superficial tenderness over an area of varying extent. This tenderness is increased by an attack (fig. 45), and is frequently found to extend over the occipital, sterno-mastoid and sterno-nuchal areas.

Any movement towards the erect position at once brings on an attack of pain. She cannot even lie on the left side without inducing an attack.

She stands with the feet wide apart and the knees bent. If the heels are placed together she reels and falls, more towards he right, but also backwards or forwards. Her walk is a shuffle vith the knees bent, and she cannot walk without support. All novements of the legs well and strongly performed. No paralysis.

Grasp—right 50, left 30, on an exceedingly weak dynamoieter. Considerable inco-ordination of the right arm, less in ie left. Cannot touch the tip of her nose at all easily with ight hand. No paralysis of arms or hands.

No alteration of sensation.

Knee-jerks present on both sides but somewhat diminished. No ankle clonus. Plantar reflexes brisk. Abdominal reflexes good.

Smell and taste are good. Hearing poor—watch heard 8—9 inches from both ears.

Vision—right and left can see fingers but cannot count them; can tell light from darkness.

Right eye—optic disc considerably swollen and pale, surface seen well with + 7D.; general hypermetropia + 3D.

Left eye—swelling and hypermetropia same as in right eye. Edge of disc ill-defined. Numerous roundish soft-edged dots arranged in groups between the macula and optic disc.

No ptosis or ocular paralysis. Marked nystagmus on looking to left or right. Occasionally, especially when looking down, there is a tendency to rotatory nystagmus.

Sensation of face good.

No paresis of muscles of face. Masseters act well.

Tongue comes out straight.

Palate moves fairly well.

Sphincters normal.

No cough or expectoration.

No abnormal physical signs in the chest.

Appetite is bad, but she takes her food fairly well, though quite without relish. She does not vomit except with the headache, and not always with every attack. Bowels confined.

Abdomen normal. No tenderness, superficial or deep. Lower edge of liver and spleen not felt.

Urine 1020 sp. gr.; acid; no albumen.

September 11. Attack this afternoon, in which she vomited almost immediately. During the attack the scalp was tender; but seven minutes after she vomited there were only a few remains of the tenderness in the occipital region. She says the pain may last for twenty-four hours if she does not vomit. Pulse becomes very feeble in these attacks. Respiration 40.

September 18. She died in an attack.

Post-mortem.—The brain was soft and removed with difficulty. The cerebellum was adherent on both sides to the brain. In attempting to separate them a rent was made in the upper wall of the right lobe of the cerebellum. From this rent gushed clear straw-coloured fluid. The whole of the right lobe was excavated by a large smooth-walled cavity. Only at one point was anything found suggesting new growth. This was some gelatinous looking material with blood vessels at the upper and outer corner. The brain showed no abnormality except great distension of the ventricles.

No abnormality in any other organ of the body or head, except the eyes.

In the large majority of cases I think it is impossible to say whether the rise of tension be in one part of the brain or in another. However, in most cases of cerebellar tumour the superficial tenderness lies over the occipital areas, and has a great tendency to affect the areas of the neck (third and fourth cervical). This affection of the neck is uncommon in the cases of tumour of the cerebrum. In cerebellar disease the superficial tenderness is more persistent in the occipital regions than elsewhere. It appears there earlier and disappears later than anywhere else on the scalp. Moreover, the pain has a tendency to be situated below the occipital protuberance, and hence to dart through to the fronto-nasal and mid-orbital regions, both of which may be tender. On the other hand, in the rare cases where we have reason to suppose that the rise of tension is mainly in the lateral ventricles, the referred pain lies mainly in front of a vertical line from ear to ear (Case 43). The pain is more persistent over the front of the scalp, and appears earlier and disappears later in that region than elsewhere.

When the disease affects the cerebrum it is frequently impossible to say, from the distribution of the superficial tenderness only, on which side the lesion lies. For on the hypothesis I have just set forth, though the lesion is unilateral, the cause of the tenderness and pain (pressure) is notoriously bilateral.

But by paying attention to the following points I think we may find that referred pain and superficial tenderness become of more help in localising the lesion than has been the case up to the present. (1) What is the position of the maximum tenderness, *i.e.*, where is the tenderness most acute, and on what side are most areas involved ? (2) Where does superficial tenderness first appear, and at what spot does it linger longest ?

If local pain and deep tenderness are a sign of meningitis, and referred pain with superficial tenderness point to rise of tension in the brain itself, it might be thought that it would be easy to diagnose an intra-cerebral lesion from a general (as opposed to a local) meningitis. This is not so. For a general meningitis mostly infects the base, and thus leads to rise of tension in the brain itself. Thus many cases of general meningitis suffer from marked referred pain and superficial tenderness. The cause is found after death in the great distension of the ventricles, so common in such cases.

Thus to sum up the conclusions to which we have arrived in this section :—

(1) In every case of brain disease we must first decide

whether the pain is local and associated with deep tenderness, or referred and accompanied by superficial tenderness of the scalp.

(2) Local pain and deep tenderness point to some affection of the dura mater in the region of the tender spot. This affection of the dura mater may be due to a local primary meningitis, or may be secondary to the growth of a tumour in the cortex.

(3) Referred pain and superficial tenderness point to rise of intra-cerebral pressure, due either to some destructive lesion in the substance of the brain itself, or to a general meningitis affecting the base. The other symptoms and signs exhibited by the patient make the diagnosis between these two alternatives fairly easy as a rule.

CHAPTER III.

Headaches referred from Thoracic and Abdominal Viscera.

Amongst the ancients it was well known that affections of the thoracic and abdominal viscera were the cause of many headaches. From the earliest times up to the present day, physicians have attempted to determine the position of the different headaches produced by various abdominal and thoracic diseases. Thus we have the "anæmic," "dyspeptic," "rheumatic," and a host of other headaches associated each with its own disease. One observer has gone a step further and classified certain headaches according to the drugs which relieve them, speaking of the headache relieved by acids, by alkalis, or by purgatives. In this way an enormous literature has grown up around the question of the forms of headache associated with visceral disease.

Again, for generations scalp tenderness has been recognised as the accompaniment of certain headaches, and this association finds expression in a popular German term for headache. It has, however, been looked upon more as a curiosity than as a matter for serious investigation.

It soon became evident in the course of this investigation, that the head, like the body, shewed areas of hyperalgesia in connection with the pain produced by diseases of the eye, ear, and other organs situated in the head itself. The next step was to see whether areas of tenderness on the scalp accompanied the headaches which were known to be produced by diseases of the thoracic or abdominal viscera. It soon became evident that such an association existed with a certain number of such headaches. On the other hand, some were found to be unaccompanied by tenderness of the scalp.

Thus in this section I shall only deal with those headaches produced by lesions of the thoracic and abdominal viscera, that are associated at one time or another with patches of superficial tenderness on the head or neck. I shall entirely neglect all headaches which may be due to visceral causes but are not at any time associated with superficial tenderness of the scalp.

At first I attempted to classify the areas of scalp tenderness according to the thoracic and abdominal diseases in which they occurred. But in this attempt I utterly failed. Meanwhile the material I had gathered seemed to shew that the clue lay in an association of certain areas of the scalp with certain areas of tenderness on the thorax and abdomen. Thus I many years ago associated the temporal area of tenderness on the head with tenderness of the subscapulo-ensiform (seventh dorsal) area on the chest. Further experience proved this association to be correct, and that we must no longer look upon these headaches, associated with enderness of the scalp, as "anæmic," "dyspeptic," &c., but s a representation of areas of superficial tenderness on he chest or abdomen. Thus, speaking broadly, it is quite mmaterial for the production of a temporal headache, with tenderness over the temporal area of the scalp, whether the disease be phthisis destroying the base of the lung, gastritis, mitral disease, or anæmic gastralgia. The only necessity is that any one of these diseases should produce a certain area of cutaneous tenderness on the surface of the body (seventh dorsal).

I must therefore ask pardon of my readers for neglecting the mass of observations which have accumulated around this subject from the earliest times. For in the first place I do not base my conclusions on the situation of the headache only, but on the position of the accompanying tenderness. Secondly, I do not look upon any particular disease as the determining factor in the position assumed by the headache and tenderness. But I seek to explain its regional distribution by the same factor which determines the position of the correlated pain and tenderness on the surface of the body. This factor is the position of the spinal segment in which the sensory nerves of the affected organ end. Thus the determination of the part of the organ affected and a consideration of the sensory supply of that part is more important than a statement of the general disease from which the patient is suffering. For this reason observations classified according to " dyscrasiæ " are useless for my purposes, and the statement that a headache was due to " cardiac disease " is of no help unless the exact condition of the heart be stated.

The proof that an area on the chest or abdomen is associated with a certain area on the scalp runs on three lines. Firstly, there are certain cases where a single area on the chest or abdomen is accompanied by one area only on the scalp. But, as I pointed out in the first part of this investigation, the appearance of a single isolated area of superficial tenderness on the body is a rarity, of extreme scientific interest but of uncommon occurrence. Much more, then, is it difficult to find cases in which not only one area on the body is present alone, but the appropriate headache is also present, uncomplicated and to its full extent. Howeve such conditions are occasionally fulfilled, and my experienc has enabled me to gather together a fair number of suc cases[1] in which one area on the body was associated with one area on the scalp.

The second method, which can be practised every day in the wards, is to notice the effect produced on the scalp tenderness which is already present, by the appearance of a fresh area of tenderness on the body. The want of diagrammatic accuracy of such observations is compensated by the frequency with which the opportunity occurs for making them.

---

[1] For this purpose study of patients in a hospital is absolutely essential. For isolated areas mostly occur at the beginning of some fresh disturbance, or when it has almost passed off. Out-patient practice shows us disturbances in full swing, or the results of an acute disturbance that has passed off. Patients who are suffering from an acute recrudescence of a chronic disease do not come to the hospital till the acute attack is passed.

The third and least reliable method is to watch the effect produced on the areas of the scalp by the disappearance of any area on the body.

To illustrate these three methods let us consider the headache and tenderness associated with the presence of the subumbilical area (tenth dorsal) on the body. Fig. 46 is taken from a case of cardiac failure and the presence of the subumbilical area was due to the rapid enlargement of the liver. The patient had suffered from mitral regurgitation without pain, and had been under observation for two months. The heart then failed, the liver enlarged rapidly, and she began to complain of pain below the umbilicus on

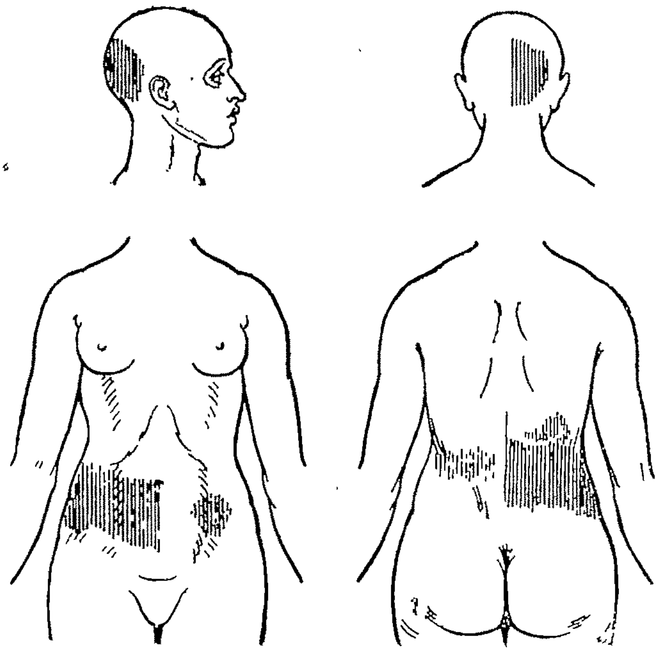


FIG. 46.

To show the association between tenderness over the tenth dorsal area on the body and the occipital area on the scalp (Case No. 46).

both sides, worse on the right side than the left. With the appearance of this area in the abdomen, she complained of occipital headache, and the scalp was found to be tender over the area in fig. 46.

Case 46.—Sarah S. (Victoria Park, Dr. Eustace Smith), aged 25. Single.

July, 1893. Acute rheumatism. Remained in bed a month and when she got up found that her breath was very short.

September, 1893. Admitted to V.P. Hospital with mitral stenosis and regurgitation.

Apex beat diffuse and forcible. Diastolic apical thrill. Upper limit cardiac dulness, third rib. Right limit, right edge of sternum. Left limit, nipple line. At apex first sound is loud and followed by a systolic murmur conducted as far as the left axilla. Second sound heard at the apex and followed by a short diastolic murmur. The pulmonary second sound was reduplicated. Aortic second sound normal.

Pulse small, irregular, easily compressible.

Edge of liver just felt.

She much improved, and was discharged on December 13, 1893.

As soon as she was discharged she tried to work about the house. Within a few days began to vomit. Much pain in the abdomen and headache.

Readmitted December 29, 1893. Condition as follows:—

Skin of face of a slightly yellowish tinge with a red spot on each cheek. Conjunctivæ not yellow. Ears and lips a little blue. No swelling of ankles. Distinct clubbing of the fingers. Knee-jerks normal. Plantar reflexes brisk.

She cannot lie flat in bed. Very short of breath on the least exertion. No fainting, but frequently feels faint.

She complains of pain in the abdomen below the navel and in the loins; she also has pain "across the shoulders and over the chest below the level of the nipples." Much superficial tenderness.

Headache over the occipital region and over both temples associated with superficial tenderness of the scalp.

Pulse about 84, irregular both in force and rhythm, small stroke, compressible.

Distinct pulsation, but no marked distension of the veins of the neck.

Apex beat in fifth space in the nipple line. Pulsation also seen in third and fourth spaces internal to nipple line. Upper limit of cardiac dulness, third rib. Right limit cardiac dulness, right edge of sternum. Left limit cardiac dulness, outside the nipple line. Heart's action very irregular. With the short beats a systolic murmur only is heard, but when the diastole is prolonged a soft diastolic murmur is heard. The first sound is feeble at the apex, but can be heard independently of the systolic murmur. The systolic murmur is conducted to the angle of the left scapula. No murmur heard over aortic area. First sound feeble over the right edge of the sternum.

Liver a good 2 inches (5 cm.) below the border of the ribs.

Suspicion of fluid in the abdomen.

Respiration 22. Slight cough; worse at night.

Some crackling sounds heard at the bases of both lungs and at apex of right lung.

Tongue red and a little furred. Bowels opened daily. She frequently vomited undigested food.

She has not menstruated for five months. Usually lasts two days. No pain.

January 9. The feet are swollen to-day and there are distinct signs of fluid in the abdomen.

The liver edge can now be felt just above the level of the umbilicus.

She complains of pain in the abdomen below the umbilicus, and occipital headache.

Marked superficial tenderness of the abdominal wall over the area in fig. 46, with definite tenderness of the occipital region of the scalp.

The liver continued to enlarge and the pulse became very irregular and small. She died on January 20, 1894.

On January 6 the temperature began to rise and oscillated irregularly between 100° and 98° F. (37·8° and 36·7° C.).

The urine was acid, sp. gr. 1025, and contained an amount of albumen which varied from about $\frac{1}{12}$ to $\frac{1}{10}$.

Post-mortem.—No pericarditis. Right side of the heart dilated. Tricuspid orifice admitted four fingers. Pulmonary valves otherwise normal. Left ventricle hypertrophied. Some thickening of the edges of the mitral valve which was somewhat stenosed. The valve was incompetent. Around its base on the auricular surface was a small layer of bead-like vegetations. The aortic valves showed fresh vegetations, which contained treptococci in considerable numbers.

No peritonitis. Fluid in the abdomen.

Liver weighed three pounds six ounces. Congested and fibrotic.

Kidneys congested, but otherwise normal.

Spleen small and congested with small fresh infarct in upper part.

Left lung healthy except for congestion at the base. Right lung congested. In middle lobe was a small infarct with some collapse around it.

Pelvic organs normal. Ovaries showed no sign of recent menstruation.

An exactly similar association between the subumbilical area and occipital headache and tenderness, is shown in fig. 7. Here the pain was due to intestinal disturbance. The case is reported on p. 356.

In the same way menstruation will frequently produce pain over one or both subumbilical areas, with tenderness in the occipital region of the scalp.

So far we have only sought cases in which the subumbilical area only was tender, and have attempted to show that it may be associated with tenderness over the occipital region. This may be called the direct method. The second, or indirect method, consists in noticing the effect produced on the tenderness already present on the scalp by the sudden addition of the subumbilical area to the tender areas on the abdomen. This sudden advent of the subumbilical area is especially well seen in cardiac cases. For certain of these cases are most difficult to keep at rest, and when allowed out of bed are liable to unduly exert themselves when not under supervision. In such cases the heart may fail sufficiently to cause enlargement of the liver, with pain and tenderness below the umbilicus. Occipital headache and tenderness will frequently then be added to the headache and tenderness, which may be already present on the scalp.

Thus we find that the occipital area on the scalp is associated with such disturbances as produce tenderness over the subumbilical or tenth dorsal area on the abdomen.

The area on the abdomen just above the umbilicus (ninth dorsal) does not commonly appear alone; but I possess a few cases showing the direct association of this area with the parietal area on the scalp. The case I have selected fo illustration was one of fibroid phthisis (fig. 6, Case 6, p 354), in which a fresh outburst took place on the left side the side opposite to the permanently damaged lung. The left side of the chest and abdomen showed the ninth dorsal fully developed, with the eighth dorsal partly present. On the right side of the chest the maxima of the ninth dorsal only are present. Corresponding to these areas on the abdomen and chest, the parietal area is beautifully shown on the left side of the scalp, with the maximum of the same area on the right side of the head.

We frequently have the opportunity of verifying this relation between the ninth dorsal area and the parietal area on the scalp by the indirect method, in those cases of phthisis where one base is becoming implicated or in those gastric cases where the ninth dorsal becomes added to the two other gastric areas. Under such circumstances we find that the tenderness of the scalp passes back behind the vertical level of the ears coincidently with the addition of the ninth dorsal area to the other areas on the abdomen and chest.

The eighth dorsal area is also one that is rarely seen without admixture with the areas around it. But its headache is a peculiarly easy one to note, and is the well-known vertical headache of many liver disorders frequently asso-

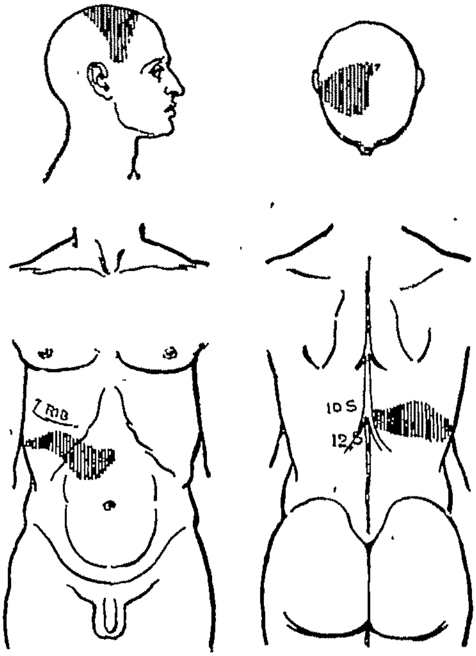


FIG. 47.

To show the association between the eighth dorsal area on the body and the vertical area on the scalp (Case No. 47).

ciated with tenderness over the vertical area of the scalp. I have selected for illustration (fig. 47) the case of a man who suffered from tubercular phthisis affecting mainly the right side, with active disturbance at the right base. He developed pain and tenderness in the right side, and on one occasion the eighth dorsal area was found to be marked out in a very beautiful manner. With this band of tenderness was associated a tender area on the scalp corresponding to the vertical area.

Case 47.—Charles M. (Victoria Park, Dr. Heron), aged 34. Admitted December 15, 1893.

He has had a cough for some time, especially in the winter. Nine weeks ago he suddenly felt ill, lost his appetite. He went to bed, felt very hot and sweated a great deal. He vomited after his meals and suffered from nausea. During this time he has wasted and suffered from pain across the lower part of the chest.

On admission his condition was as follows:—

Medium sized man of 35. Looks about his age. Lips a little pale and blue. No œdema. Marked wasting of the limbs and coverings. Profuse nocturnal sweating.

Respiration 36, regular, not short of breath as he lies in bed, but short of breath on the least exertion. Cough not very troublesome. Expectoration greenish-yellow. Semi-fluid containing a quantity of mucus. Tubercle bacilli found.

The pain has now passed off, and he now has no headache or tenderness of the scalp.

The movement of the chest is bad. No dulness over apices in front, but the note is markedly deficient over both upper lobes behind as low as about the level of the sixth vertebral spine. At the right apex in front, the breath sounds are just heard and vocal resonance is diminished; râles heard after coughing. At the left apex in front the breath sounds are scarcely audible and vocal resonance is nearly absent; a few râles are heard after coughing. Over the right lung behind the breath sounds are hollow and the vocal resonance is increased over the extreme apex, whilst at the level of the spine of the scapula, the breath sounds are cavernous and whispered pectoriloquy is heard. A similar condition obtains over the upper part of the left lung. From the angle of the scapula downwards, on both sides the breath sounds and vocal resonance are feeble, but no râles are heard.

Pulse 100, regular, poor stroke. Artery distinctly hard for a man of 35.

Cardiac dulness is masked. Heart sounds normal. No murmur.

Tongue clean, moist, a little pale. Appetite now fair. He vomits with his cough, but not otherwise.

Liver not felt. No abnormal signs in the abdomen.

Urine 1020 sp. gr., acid. No albumen or sugar.

February 23. Yesterday he began to complain of pain over the right costal border and pain over the right vertex. To-day there is definite superficial tenderness both of the body and scalp as in fig. 47.

His cough is about as before. He is sweating more at night.

The signs at the left apex are quieter and fewer râles are heard.

On the other hand there is now diminution of percussion note over the right base. Below the angle of the right scapula, the breath sounds are very weak, but vocal resonance is increased and numerous râles are heard all over the right base below the angle of the scapula. The left base is free as before. No friction heard anywhere.

Since his admission he has suffered from occasional slight attacks of diarrhœa. The motions became loose two days ago. The pain over the eighth dorsal area does not seem to be affected by the passage of the fluid motions. He now has three motions in twenty-four hours.

His temperature has been irregular and high from the beginning, sometimes touching 102° F. (38·9° C.), but generally reaching the normal in the morning.

The seventh dorsal is one of the easiest areas to associate with its proper headache, owing to the frequency with which the two areas of tenderness appear together. Fig. 48, chosen to illustrate the relation of this area to the temporal area of the head, is taken from a case of anæmia with gastralgia.

Case 48.—Letitia A. (Victoria Park Chest Hospital, Dr. Thorowgood), aged 28, single, cook. Admitted April 24, 1894.

For a long while she has suffered from pain in the left side of the chest and back.

For the last four winters has been troubled with cough. No cough in the summer.

Christmas, 1893, had her usual winter cough, which ceased in January, 1894.

For six months she has been getting thinner, and also steadily paler.

Short of breath for twelve months. Worse the last three months.

No acute rheumatism.

Menstruation began at 13. Regular every four weeks; lasts seven days. Six months ago she would use two diapers on the first three or four days. For the last three months has used scarcely one a day. The discharge is paler than it used to be. She has had no pain on menstruation lately. No vaginal discharge.

No abnormality in her family history.

On admission:—

Medium sized woman of 28. Face very pale. Forehead and skin of body pale, with a faint yellowish tinge. Lips and orbital conjunctivæ pale. Ankles swell when she walks about. Wasting of neck, upper part of chest and of muscles of extremities. Knee-jerks decidedly increased; equal. No ankle clonus. Plantars obtained with difficulty.

No fainting. No palpitation except on exertion. Very short of breath when she goes up an incline, or upstairs.

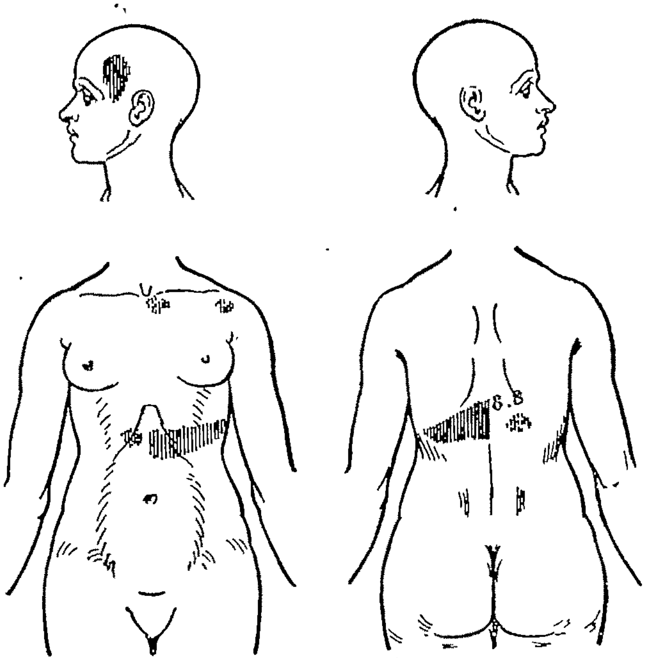


Fig. 48.

To show the association between the seventh dorsal area on the body and the temporal area on the scalp (Case No. 48).

She complains of pain over left lower ribs, in epigastrium and in the back below the angle of the left scapula. It comes on if she has been walking or lifting anything heavy. The pain disappears when she is in bed. Very seldom in right side; practically only in left side.

Headache in left temple. It starts as a very sharp pain in the temple, lasting a short time and followed by a dull aching pain.

Marked and well-defined superficial tenderness on the body and left side of the head over the areas shown in fig. 48.

Pulse 82, regular. Fair stroke. No pulsation in veins of neck. Bruit heard in veins of neck on right side.

Estimation of the blood:—Hæmoglobin 48 per cent. (by Gowers' hæmoglobinometer). Corpuscles 93·8 per cent., or 469 in 10 squares (by Gowers' hæmacytometer).

Apex beat fourth space internal to nipple line. No thrill. Upper limit cardiac dulness, lower border third rib. Right limit, left edge of sternum. Left limit, one inch internal to nipple line. A soft systolic murmur heard over the apex beat, but not conducted with the axilla or upwards. The first sound over the apex is not obscured by the murmur. Accentuated pulmonary second sound.

Respiration 20, regular. No cough. No expectoration.

Movement of right apex a little better than that of left. Percussion note perhaps a little diminished under left clavicle. Distinctly diminished over extreme left apex behind. Over left apex in front breath sounds are harsh and expiration is a little prolonged; vocal resonance not increased. At left apex behind, breath sounds are weak, vocal resonance is diminished, and occasional rhonchus is audible. Elsewhere signs normal.

Tongue moist and a little grey. Appetite variable. Sometimes fairly good. At other times she cannot eat anything.

Sometimes, if she has no pain, food will cause the pain to appear over the pit of the stomach and in the back. If the pain is already present taking food makes it worse. The pain comes on directly she eats or ten minutes after taking food, and if it comes on in the middle of the day (after dinner) it will last till the next morning. If she lies in bed the same diet will hardly cause any pain. If she is up and about, the same food will cause a great increase in the pain that is always present when she is up. No vomiting. Frequent nausea. Bowels open once daily as a rule, without purgatives. Liver and spleen not felt. No physical signs in abdomen.

Urine, specific gravity 1042; acid. No albumen or sugar.

Temperature normal.

As we pass higher up on the chest the areas become in themselves more difficult to mark out, and their correlation with areas on the scalp more difficult to determine. For the third, fourth, fifth and sixth dorsal areas all tend to appear in combination, and if they are present singly they are, as a rule, not fully developed, but represented by their maxima only.

The area of the sixth dorsal is not uncommonly affected in consequence of cardiac lesions, either with or without the seventh dorsal. It is associated with the fronto-temporal area on the scalp. The case selected for illustration is one of aortic stenosis, and shows the relation between these two areas very beautifully (fig. 49).

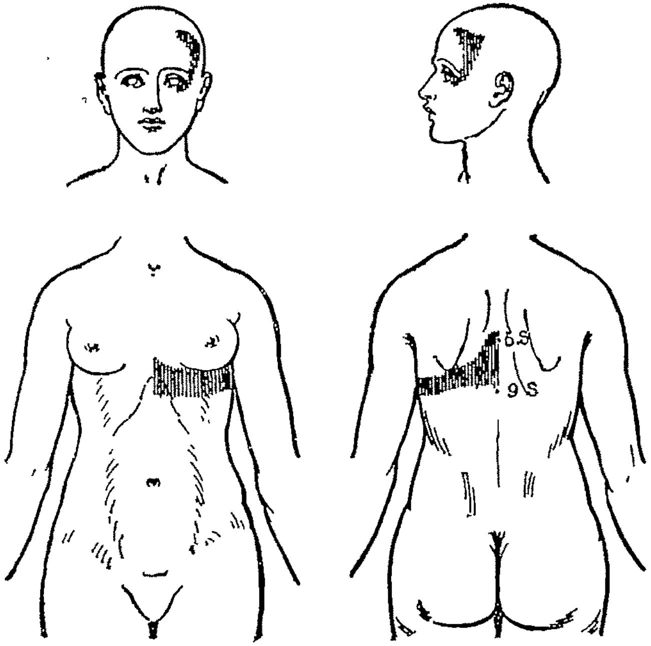


FIG. 49.

To show the association between the sixth dorsal area on the body and the fronto-temporal area on the scalp (Case No. 49).

Case 49.—Elizabeth L. (Victoria Park, Dr. Smith), aged 17. At the age of 8 years had "rheumatism." Legs, hands, and elbows swelled. At age of 15 had a second attack. Feet, hands and elbows again swelled, and "little growths" came on the elbows and knuckles. Has never been quite well since. Was admitted to V.P. Hospital for morbus cordis, May, 1893. Got worse directly she left the hospital. Much pain. Gradually improved, and was fairly well till December, 1893. Then began to suffer from pain and "irregular action of the heart."

Re-admitted, March 2, 1894. Condition as follows :—

Medium-sized girl of 17. Well-developed ; looks much older. Face of a uniform pinkish colour. No cyanosis. No flush on cheeks. No wasting. Knee-jerks normal. Plantar reflexes good. No fainting. Palpitation on the least exertion. Heart beats irregularly. Can walk slowly on level ground, but when she goes upstairs has a feeling of suffocation as if she could not get her breath. Heart thumps against the chest and she feels faint.

She always has pain to a greater or less extent under the left breast going through to the angle of the scapula behind. At other times she has pain over the upper part of the chest on the left side and in the left arm to the elbow.

Troublesome headache "in the temples."

Pulse 80, regular, small stroke; not a regurgitant pulse. No pulsation of the arteries or veins of the neck.

Apex beat in fifth space one and a-half inches (4 cm.), outside nipple line. Upper limit of cardiac dulness second space. Right limit, close to right edge of sternum. Left limit, left nipple line. Over the right base a systolic murmur is heard conducted over the sternum and heard over the apex beat. The murmur over the apex beat does not obscure the first sound and is probably the same as that over the right base. Over the lower part of the sternum a slight but distinct diastolic murmur is heard. The systolic murmur is not heard in the left axilla or at the angle of the left scapula. Diastolic murmur not heard at the apex. Sounds on right side of heart good.

Liver not enlarged. No œdema or ascites.

Respiration 20. No cough. No abnormal physical signs in the lungs.

Urine sp. gr. 1018. No albumen.

When admitted she was in the third day of menstruation and thus the pain and tenderness were wider spread than usual. She soon settled down to the usual pain under the left breast and upper part of left side of chest and arm associated with superficial tenderness.

March 27. The pain and tenderness in the body and head has been steadily diminishing under the influence of rest in bed, and to-day the only remains consist of very slight pain under the left breast associated with a band of tenderness and fronto-temporal headache accompanied by tenderness over the fronto-temporal area of the scalp (fig. 49).

The pulse is 70, and the heart's action is quieter, but otherwise the signs remain as before.

Finally all pain and tenderness disappeared, but reappeared for a short time after she had attempted to run upstairs

The eyes were normal. Nothing in disc, fundus, or media. Vision: right eye, $\frac{6}{9}$; left eye, $\frac{6}{6}$; Jager 1 in both. Hearing, smell, taste, good. Nothing abnormal in ears, nose, or pharynx.

Temperature throughout normal. Highest 99 6°, on one occasion.

The relation of the sixth dorsal area to the fronto-temporal area of the scalp is best seen by the indirect method. Thus we repeatedly find that the addition of the sixth dorsal area to the seventh dorsal adds the fronto-temporal to the temporal area previously present on the scalp. In a case of mitral obstruction and regurgitation it was interesting to see how the fronto-temporal area faded away as the sixth dorsal diminished, whilst at the same time the temporal area increased as the seventh dorsal became more evident.[1]

The fifth dorsal area is exceedingly difficult to obtain pure, and in several of my cases in which it has appeared alone, the headache was not developed or only faintly marked. This area on the body seems, however, to be associated with the fronto-temporal area on the head. In Case 50 (fig. 50) the fifth dorsal area was present on the left side of the chest,

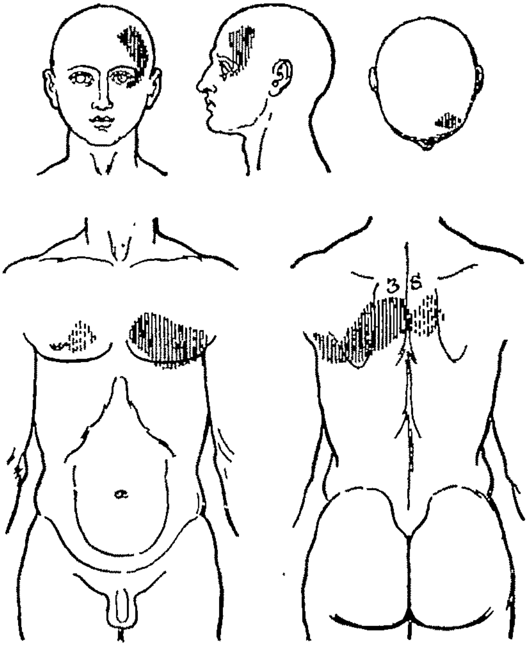


FIG. 50.

To show the association between what I take to be the fifth dorsal area on the body and the fronto-temporal area on the scalp (Case No. 50).

---

[1] This case will be published in full in my paper on "Mental States in Visceral Disease."

and was associated with the fronto-temporal area on the left side of the forehead.

By the indirect method we repeatedly see hyperalgesic areas with the characteristic upper border of the fifth dorsal, associated with tenderness on the head, which is bounded in front by the equally characteristic anterior border of the fronto-temporal area.

For the first time, in passing from below upwards, we have come to two areas on the chest, which are associated with the same area on the scalp—a condition which we shall find repeated in all the areas above this level on the chest.

Case 50.—Edward S. (Victoria Park Chest Hospital, Dr. Thorowgood), aged 36. Married. Tea porter. Admitted May 2, 1894.

Cough every winter for the last six to seven years. It comes on as soon as the bad weather sets in, and passes off with the winter.

January, 1893. Had a bad attack of "bronchitis," and has never been well since. Has had a cough and has spat phlegm ever since.

Since January, 1894, he has sweated at night and lost much flesh.

No hæmoptysis.

History of phthisis in the father's family, but not amongst brothers and sisters.

On admission :—

Tall man of 36. Face and lips a fair colour. No anæmia. No œdema of face or ankles. No cyanosis. No clubbing of fingers. Some wasting, showing itself mainly in the diminution in his well-developed muscles. Knee-jerk and plantar reflexes normal.

Respiration 18. Short of breath on exertion. Not as he lies in bed. Cough troublesome at night. Very little expectoration (no tubercle bacilli could be found on two examinations).

He complains of pain in the left nipple. Sometimes this goes through to his shoulders, and hurts him when he breathes or coughs. Marked superficial tenderness as in fig. 50.

He says: "I suffer from no headache, but from constant soreness of the scalp." He cannot put his hat on, his head is so tender. Marked superficial tenderness as in fig. 50.

At the apices the movement is about equal, but the right base moves better than the left. In front there is a little diminution of percussion note under right clavicle, but behind the note is distinctly diminished over the left apex and mid-scapular region. Breath sounds and vocal resonance fair under both clavicles. A few râles at both apices. Behind breath sounds fair, but expiration prolonged and vocal resonance diminished over left apex. In left mid-scapular region expiration prolonged and vocal resonance increased. At left base breath sounds fair and vocal resonance about normal. A few râles are heard all down the left back, especially about angle of left scapula. Not at extreme left base. No friction anywhere.

Pharynx somewhat red. Larynx absolutely normal.

Pulse 60, regular, good tension.

Physical signs in the heart normal.

During his stay in the hospital he was frequently troubled with pain and superficial tenderness over a variable area in the left side of the chest, mainly in the region of the fifth and sixth dorsal. Frequent fronto-temporal headache.

When he left hospital pain was improved and chest was much freer of râles. Other physical signs as before.

His temperature was never higher than 99·4° F. (37·4° C.) He gained 7½ lbs. in six weeks.

It is difficult to decide what tenderness of the scalp is associated with tenderness over the fourth dorsal area on the body. My observations give apparently contradictory results, for sometimes I find this area apparently associated with the fronto-temporal, at other times with the mid-orbital area of the scalp. I must, therefore, leave undecided whether the fourth dorsal area on the body is associated with the fronto-temporal or mid-orbital area on the scalp.

The third dorsal area appears to be definitely associated with the mid-orbital area on the scalp (fig. 51).

Case 51.—Margaret R. (Victoria Park Chest Hospital, Dr. V. D. Harris), aged 21, single, dressmaker. Admitted March 20; died April 15, 1894.

She has always been "delicate" and subject to coughs and colds.

Twelve months ago she had a sore throat; since then she has had a constant cough.

September, 1893. She began to spit up small quantities of fluid blood; phlegm remained streaked for some time afterwards. Has frequently had slight hæmoptysis since then.

Summer 1893, attacks of diarrhœa. Attacks on and off ever since.

Has sweated at night for the last six months.

Marked family history of tubercular disease (mother).

On admission:—

Face long and thin. Complexion of a chalky whiteness, with a slight pink flush on the cheeks. No cyanosis. No œdema of face or ankles. Extreme general wasting. Marked sweating. Knee-jerk and plantar reflexes good.

Respiration about 40. Very short of breath even when lying in bed. Cannot walk more than a few steps. She becomes extremely short of breath after coughing.

Cough frequent. Small groups of single coughs. Not paroxysmal.

Small quantity of irregular masses of muco-purulent expectoration.

She complains of pain in the upper part of right axilla, in the right shoulder and upper edge of right shoulder blade, also in right side of abdomen below the umbilicus and in the right loin.

Headache over anterior part of temples, in the temporal region, and over the vertex.

Marked superficial tenderness of chest and back. Tenderness of scalp.

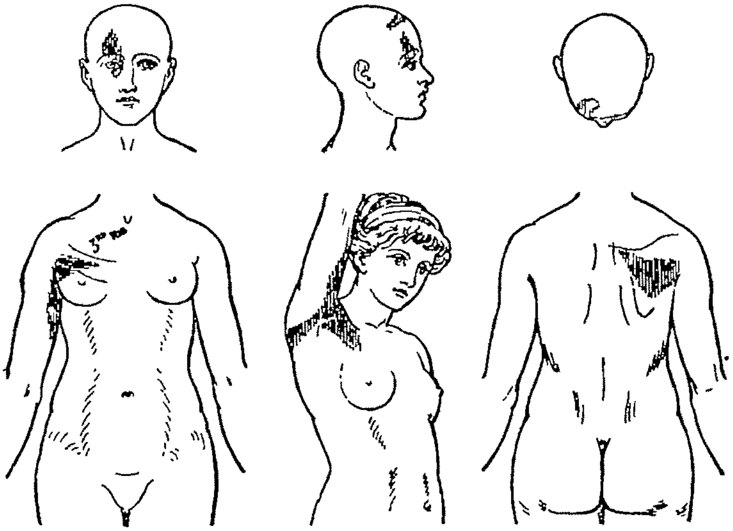


FIG. 51.

To show the association between the third dorsal area on the body and the mid-orbital area on the head (Case No. 51).

Movement of left apex greater than right. Diminished percussion note over apices behind and over both mid-scapular fossæ as low as angle of scapulæ on both sides. Cavernous breathing and whispered pectoriloquy over upper part of right lung in front as low as fourth space. Cavernous breathing and whispered pectoriloquy over a more limited area at left apex in front. Behind cavernous breathing and whispered pectoriloquy at both apices as low as level of fourth dorsal spine. Râles over front of chest both sides. Behind, râles as low as angle of scapula on left side, and to base on right side. No friction heard anywhere. Pulse 120, low tension, regular.

No abnormal physical signs in heart.

Tongue clean, moist, of a fair colour. Appetite good. No vomiting. No diarrhœa now.

Urine sp. gr. 1020; acid. No albumen, no sugar.

The pain and superficial tenderness was usually wide spread, but on April 1 she had less pain, and the tenderness happened to lie only over the areas in fig. 51. This in no way represents her usual pain and tenderness, which was wide spread as a rule.

Vision: right eye; $\frac{6}{12}$, Jæger 1. Left eye, $\frac{6}{18}$; Jæger 1.

Disc and fundus oculi normal. Slight myopia (about 1D. in the left eye.

The temperature ranged between 102° at night and about 98° to 99° in the morning (about 39° to 36·7° C.).

She gradually became worse and died on April 15.

The second dorsal area, with its characteristic flap on the inner side of the arm, seems to be associated with the mid-orbital area of the scalp. This association is fairly easily determined owing to the prevalence of the second dorsal area in certain forms of aortic disease.

Thus, at least two areas on the chest are more or less associated with tenderness over the mid-orbital region of the scalp. But before this relation can be established each case must be carefully examined for hypermetropia. For, as I pointed out on p. 395, hypermetropia is peculiarly associated with tenderness over the mid-orbital area of the scalp. Case 52 has been chosen to show the method of procedure in such cases, and to illustrate the reasons which led me to think that the headache and tenderness of the forehead were due to the aortic disease, and not to the hypermetropia. First of all, the headache and tenderness were determined, and then, without in any way altering the daily length of time for which she was allowed out of bed, the eyes were put heavily under atropine. At the end of four days the headache was no better. Then she was kept strictly in bed, and the headache and tenderness disappeared. When she was again allowed up headache and tenderness reappeared, but again disappeared when she was put to bed. Thus, in this case, I think the mid-orbital headache and tenderness was associated with the cardiac condition (producing pain over the second dorsal) rather than with the hypermetropia.

I possess other cases illustrating this association between the second dorsal area on the body, and the mid-orbital on the forehead, in which hypermetropia was absent, but I have quoted this instance to illustrate an attempt at differential diagnosis in such cases.

When the mid-orbital area on the forehead is tender from some thoracic disease, the patient will complain that reading or sewing is painful, although hypermetropia is absent. Thus, at one time a patient suffering from pulmonary or cardiac disease will be unable to do near work without pain, whilst within a short time the headache may have shifted its position, and no longer occupy the mid-orbital area, and reading or near work will no longer increase the headache.

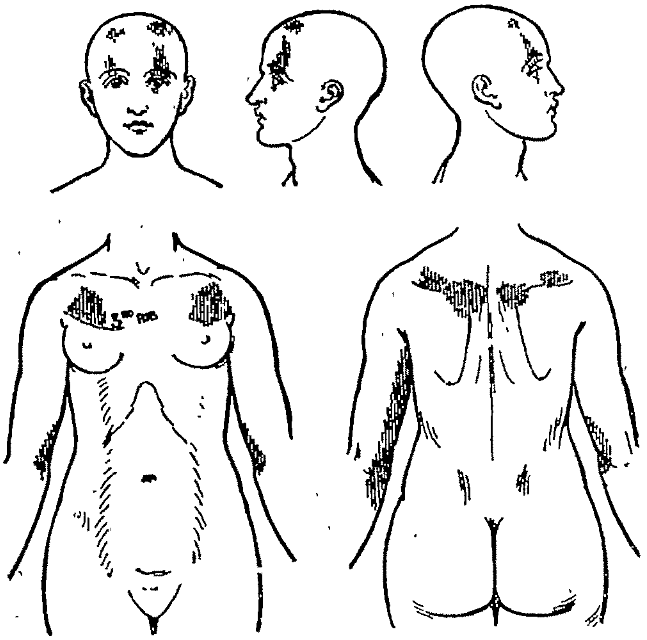


Fig. 52.
To show the association between the second dorsal area on the body and the mid-orbital area on the scalp (Case No. 52).

Case 52.—Edith B. (Victoria Park Chest Hospital, Dr. Heron), aged 13.

Admitted March 20; discharged May 22, 1894.

Just before Christmas, 1893, she had an attack of rheumatic fever. Ankles and knees were swollen and painful, wrists swollen but fingers were not affected.

Since this attack she has been short of breath, and has suffered from pain in the upper part of the chest with bad headache over the eyes.

Nothing of importance in the past or family history.

On admission:—

Small girl of 13, exceedingly intelligent and bright. Lips a fair colour. No cyanosis; no anæmia; no wasting. Knee-jerks and plantar reflexes good.

No fainting. Palpitation on exertion, which is accompanied by pain. Very short of breath on exertion but not when quiet or in bed.

She complains of pain over the upper part of the chest which goes down the left arm to the elbow.

Headache over the eyes and temporal part of the forehead on both sides, worse on left than right side.

Marked superficial tenderness both of chest and scalp.

Pulse 80, regular, medium stroke; fair tension between the beats. No pulsation either of arteries or veins of the neck.

Heart's apex beat in fifth space one inch outside nipple line. No thrill. Upper limit cardiac dulness in second space. Right limit, right edge of sternum. Left limit as far out as half an-inch to left of left nipple line. Over the apex the first sound is short and sharp, and is followed by a soft high-pitched systolic murmur. This murmur is heard at the angle of the left scapula. The second sound at the apex is sometimes reduplicated, and a distinct rough pre-systolic murmur is occasionally audible. Over the right base the first sound is audible, but second sound is a little weak and thin. Over the right edge of the sternum and over the second right space a systolic murmur is heard, differing in tone from that at the apex. It is apparently distinctl separated by an interval from the apical systolic murmur. Th pulmonary second sound is accentuated over a wide area. Th first sound to the right of the lower part of the sternum is long and low pitched.

No cough. No expectoration.

No abnormal physical signs in the lungs.

Tongue moist, papillæ a little prominent. Appetite poor. Occasional nausea. No vomiting. No pain after food. Bowels confined.

Liver and spleen not enlarged. No signs of fluid in the abdomen.

Vision: right eye, $\frac{6}{9}$; Jæger 2, badly. Left eye, $\frac{6}{24}$; Jæger 3.

Hypermetropia in right eye, + 2D. in one meridian; + 2·5D.

in the other. Left eye + 2·5D. in one meridian; + 3D. in the other.

She was put strictly to bed, and the pain and tenderness on the chest and arm practically disappeared. The tenderness on the scalp diminished to a small spot over the left eyebrow.

April 10. She was allowed up and romped and played until she was forced to cease owing to the reappearance of pain. Tenderness had returned in the evening over the areas of the second and third dorsal, including the ulnar side of the arm.

The apex beat is at least half-an-inch outside the nipple line, and forcible.

April 11. The pain is nearly gone, and the tenderness is reduced to two small faint patches, one situated over the second space, and the other over the vertebral border of the spine of the scapula.

The apex beat is now in the nipple line, half-an-inch internal to the spot marked permanently on the skin last night.

She then remained in bed till April 15. Dr. Carter then allowed her to get up, after making careful observations on the condition of the pain and tenderness, and of the physical signs.

The result (fig. 52) was identical with that on April 10.

From this time she was allowed to get up for part of each day, but her eyes were put deeply under atropine. Vision then became in right eye, $\frac{6}{60}$; left eye, $\frac{6}{60}$. The estimated error of refraction rose in the right eye to + 4D., in the left eye to + 6D. all round.

The headache and tenderness of the scalp remained quite unaltered.

May 1. She was again put back to bed, and all pain and tenderness both in the body and head disappeared.

She then remained in bed for thirteen days, and during this time headache over the mid-orbital region was absent.

The first dorsal area is very rarely present except in affections of the aorta, such as aneurism. In these cases it is not associated with any headache or scalp tenderness.

We have now reached that part of the arm which does not become directly tender in visceral disease (the upper gap).

Above the gap there are two areas, the third and fourth cervical (sterno-mastoid and sterno-nuchal) which are associated with tenderness on the head. Pain and tenderness over either of these areas is associated with pain and tenderness over the fronto-nasal area. Thus, if the third cervical (sterno-mastoid) be affected the patient complains that the pain shoots from the back, through the head to the centre of the forehead (*cf.* p. 433). He places his hand just below the occipital protuberance, and then points to the centre of the forehead just above the root of the nose.

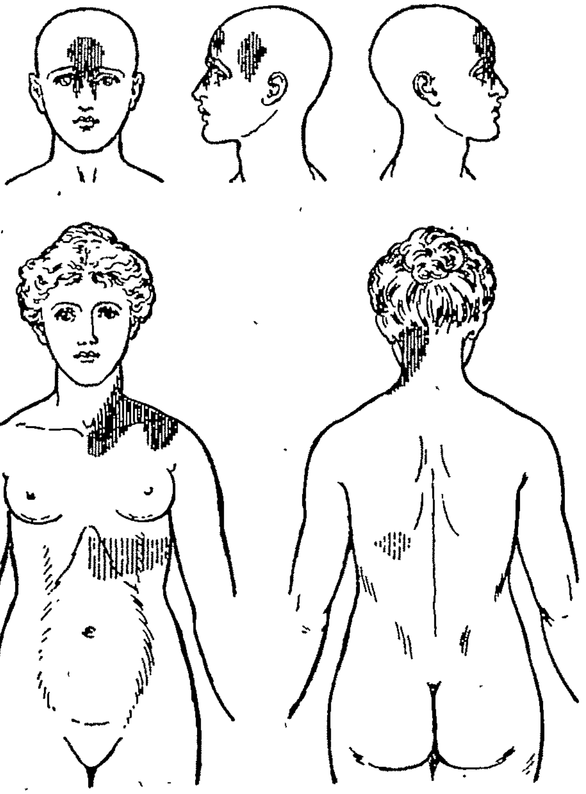


FIG. 58.

To show the association between the third cervical area (somatic) and fronto-nasal area of the forehead (Case No. 58).

This figure also shows the relation between the seventh dorsal area on the body and the temporal area on the scalp (*cf.* fig. 48).

Case 53.—Louisa S. (Victoria Park Chest Hospital, Dr. Eustace Smith), aged 29, married. Admitted April 17, 1894.

At the age of 7 had "rheumatic fever," and was in St. Bartholomew's Hospital for three months.

Since then she has had frequent attacks of acute rheumatism, the last of which was in May, 1893.

Ever since an attack of rheumatism at the age of 18 she has been short of breath, and suffered from more or less pain "under the left breast."

Since she was a girl she has been subject to "fits." Sometimes she has been free of them for years, at other times they occurred once or more in the week. She says they are always present "when her heart is bad."

She had no fits during her stay of six weeks in the hospital.

On admission :—

Medium sized woman of 29. Cheeks a little blue. Lips red with a slightly blue tinge. No œdema of face or ankles. No wasting.

No fainting. Palpitation on the least exertion, followed by pain under left breast; very short of breath on exertion.

Marked pain at epigastrium and under left breast. Headache over temples; marked superficial tenderness.

Pulse 64, regular. The artery is distinctly full between the beats. Arterial wall not hard. No pulsation in the neck.

Apex beat in fifth space just internal to left nipple line. Not heaving. Marked long diastolic thrill. Upper limit cardiac dulness, lower border third rib. Right limit, left edge of sternum. Left limit, slightly internal to left nipple line. Over the apex the first sound is short and sharp. It is followed by a systolic murmur which is not heard at the angle of the left scapula. The second sound is good. After the second sound comes a long trickling diastolic murmur; then, under ordinary circumstances, a little roughness may be heard before the first sound, but nothing beyond. If, however, she is made to move about, a rough, churning, pre-systolic murmur is heard running up to the first sound. Over the left base the second sound is exceedingly sharp and slapping. Over the right base the sounds are good. Over the right edge of the lower part of the sternum the first sound is weak, but the second sound is sharp.

Respiration 20. Very slight cough. No expectoration.

A few râles are heard at the bases of both lungs, especially of the left.

Tongue clean. Appetite fair. Pain directly after food at the epigastrium. Occasionally vomits. Pain relieved by vomiting. Bowels confined.

Liver can just be felt below the costal margin. Spleen not felt. No signs of fluid in the abdomen.

Motion normal.

Sensation normal except for the tenderness above-mentioned.

Cranial nerves normal. Pupils react to light and accommodation.

Knee-jerks equal. No ankle clonus. Plantar reflexes good.

Vision, right eye, $\frac{6}{36}$; Jæger 2, far point 4½ inches; left eye, $\frac{6}{12}$; Jæger 1, far point 10½ inches.

Considerable myopic astigmatism in the right eye. Left eye nearly emmetropic.

Disc and fundus normal.

Hearing good. No abnormality in ears.

Smell good. No abnormality in nose.

Taste good. No abnormality in pharynx or larynx.

Temperature remained about 99°.

Urine sp. gr. 1020; acid. No albumen. No sugar.

April 20. She was suffering from pain in the left side of the neck and at the epigastrium and "under left breast."

Headache "over the front of the forehead" and in the left temple. She says the pain shoots through from the back of the neck to the front of the forehead.

Definite superficial tenderness over the areas in fig. 53.

She got up yesterday for the first time.

Tongue clean. No nausea. No vomiting.

Pulse 70, regular.

Physical signs in the heart as above.

Above the third cervical (sterno-mastoid) area there is no association between the lateral areas of the head and neck, and tenderness of the scalp or forehead. Thus, neither the superior laryngeal nor hyoid areas, nor any areas of the same series are directly associated with pain or tenderness in the region of the scalp or forehead.

I began to describe the relation between areas on the body and areas on the scalp, by pointing out that the presence of the tenth dorsal was frequently associated with the presence of the occipital area on the head. When we pass below the tenth dorsal to those cases in which the pain and tenderness is situated over the eleventh dorsal or any lower area, we no longer find that the pain and tenderness on the body is accompanied directly by pain and tenderness in the head. Thus, in the following case (Case 54) of malignant disease of the cervix, although the eleventh and twelfth dorsal areas were tender, and in spite of the fact that the patient was far advanced in phthisis, she remained free from referred headache and tenderness of the scalp to the end. Again, I watched a case of salpingitis for six weeks, and although she suffered from marked pain and tenderness over the eleventh and twelfth dorsal areas, she did not complain of headache, and no tender areas were present on the scalp. Thus I believe that the abdominal organs (such as the ovaries, testes, liver and greater part of the midgut), which refer on to the area of the tenth dorsal segment, are associated with referred pain and superficial tenderness on the scalp. On the other hand, the pelvic viscera (Fallopian tubes, uterus and bladder), which refer on to the eleventh dorsal area or on to areas below it, are not directly associated with any referred headache or scalp tenderness.

I must here warn my readers from supposing that they will never find referred headache and tenderness in cases where the uterus or Fallopian tubes are at fault. For as is well-known[1] such cases are peculiarly liable to the development of pain and tenderness elsewhere than over the areas directly in connection with the organs themselves. Cases of cervical catarrh or erosion frequently develop gastralgia with pain after food, occasionally accompanied by vomiting. Under such circumstances headache and scalp tenderness may appear but will represent the gastric areas of tenderness, and obey the rules I have laid down above. Thus, if the original pain and tenderness lie below the level of the tenth dorsal area, any headache and scalp tenderness that may be present will not be correlated with the primary tender areas but with some secondary areas above the lower limit of the tenth dorsal.

Case 54.—Jessie B. (Victoria Park Chest Hospital, Dr. Heron), aged 23, married. Admitted January 17, died January 28, 1894.

February, 1892. "Fell" with her second child. Had attack of "bronchitis," followed by persistent cough, sweating and loss of flesh.

September 6, 1892. Confined. Fifteen days afterwards fainted. Went to bed for a month. Cough. Much wasting and sweating.

Began to bring up blood twelve months ago, and has had slight hæmoptysis many times since.

On admission :—

Much wasted. Face pale and wasted, with occasional brilliant red flush on both cheeks. Ears blue. Profuse sweating at night.

Respiration 40. Very short of breath as she lies in bed. Cough very troublesome. Large quantity of muco-purulent expectoration in nummulated masses. On the night of admission it contained a small amount of blood.

No pain or tenderness of any area above the umbilicus.

---

[1] *Cf.* Part i. of this work—BRAIN, 1893, p. 106.

Marked superficial tenderness just above Poupart's ligament on the left side, and in the femoral triangle. Tenderness behind, over the lower lumbar region. This tenderness marked out the areas of the eleventh and twelfth dorsal segments on the left side, with the maxima of the eleventh dorsal on the right side.

No headache. No tenderness of the scalp.

Signs of extreme cavitation over the whole of the upper part of the right lung. The right side is dull from apex to base. Over the left lung bronchial breathing and bronchophony were heard everywhere, with occasional small areas of tubular breathing, and whispered pectoriloquy. Râles all over the chest.

No abnormal physical signs in the heart.

Tongue furred and dry. Appetite fair. Bowels irregular. No pain after food. No vomiting.

Liver and spleen not felt. Nothing abnormal felt in the abdomen.

No vaginal discharge now. She has not menstruated for many months. Menstruation began at 13. Regular, with an interval of three weeks. Lasted as a rule six days. About two to three diapers daily. Much pain in the back for a day or two before, and during the flow. First child, December, 1890. Nine hours in labour. Instruments used. Milk came in on third day. Got up on tenth day. Suckled child for eleven months. Second child, September, 1892 (*Vide supra*). Occasional yellowish discharge. No blood-stained discharge apart from menstruation.

On examination *per vaginam* a mass is felt in the cervix, either an erosion or a growth. Uterus not enlarged.

Temperature sometimes reached 102° and never fell to normal.

Urine sp. gr. 1030, acid. No albumen or sugar.

At first she much improved, and was bright and cheerful. Pain and some tenderness over the areas of the eleventh and twelfth dorsal was frequently present.

On January 28 she got suddenly worse, and died in a few hours.

Post-mortem.—Right lung: Whole upper lobe one large cavity, containing pus and *débris*; lower and middle lobes much scattered tubercle. Fibrosis. Left lung: Much fibrosis of both lobes. Scattered tubercle and two small cavities.

No ulceration of the intestines, no peritonitis.

Tubes and ovaries normal.

Around the cervix and in its substance is a growth. Mucous membrane raised, roughened and in part ulcerated. The growth affects the lower end of the cervical canal. Microscopical examination showed involution cells (cancer).

Within the area between the third cervical (sterno-mastoid) and the tenth dorsal segments, pain and superficial tenderness may occur without headache or scalp tenderness. But such pain and tenderness is usually transient or slight. Should the tenderness persist for any length of time or be severe, the proper scalp tenderness will sooner or later make its appearance. As the visceral disturbance dies away the headache and scalp tenderness usually disappear before that on the body, just as it tends as a rule to appear later than the somatic tenderness with which it is associated. In a few cases, however, the headache and scalp tenderness may be in excess of the pain and tenderness on the body. I am unable as yet to say on what this depends.

The maximum tenderness on the scalp, in connection with tenderness over the dorsal areas, is on the same side as the tender area on the body. It is, however, not uncommon to find a tender area on one side only of the body with not only tenderness on the same side of the scalp, but also some less marked tenderness over the same area on the opposite side of the head. Thus, a visceral lesion on one side of the body seems to more readily cause bilateral tenderness on the scalp than on the body. The tenderness on the opposite side to the lesion comes later, disappears earlier, and is less fully developed than that on the same side as the lesion.

Before concluding this section I must allude to certain peculiarities of the third and fourth cervical areas (sterno-mastoid and sterno-nuchal). That they should become tender in diseases of the apex of the lung is not to be wondered at. But these areas also bear a curious relation to affections of certain abdominal viscera, notably to the stomach and liver. In certain diseases of the stomach we not only find that one or more of the gastric areas (seventh, eighth, or ninth dorsal) become tender, but that frequently one or other of these two cervical areas is tender in addition. Under such circumstances there may be temporal tenderness accompanying the dorsal area, and frontal tenderness accompanying the cervical area (fig. 53, Case 53). A similar condition frequently obtains in various disturbances of the liver.

I have expressed the conclusions to which we have arrived in this chapter in the following table.

Area on Body	Associated Area on Scalp.	Organs in particular relation with these areas.
Cerv. 3	Fronto-nasal (? rostral)	Apices of lungs. Stomach. Liver. Aortic orifice (?)
Cerv. 4	Fronto nasal	
Dorsal 2	Mid-orbital	Lung. Heart (ventricles). Ascending arch of aorta.
Dorsal 3	Mid-orbital	Lung. Heart (ventricles). Arch of aorta.
Dorsal 4	Doubtful	Lung.
Dorsal 5	Fronto-temporal	Lung. Heart (occasionally).
Dorsal 6	Fronto-temporal	Lower lobe of lungs. Heart (auricles).
Dorsal 7	Temporal	Bases of lungs. Heart (auricles). Stomach (cardiac).
Dorsal 8	Vertical	Stomach. Liver. Upper part of small intestine.
Dorsal 9	Parietal	Stomach (pyloric end). Upper part of small intestine.
Dorsal 10	Occipital	Liver. Intestine. Ovaries. Testes.
Dorsal 11	0	Intestine. Fallopian tubes. Uterus. Bladder (contraction).
Dorsal 12	0	Intestine (colon). Uterus, &c.

I am aware that the statements I have made in this chapter will probably have to undergo considerable amendment in course of time. I hope, however, that the main outlines of the relation between certain referred pains in the body, and certain referred headaches may be found to be correctly stated in this chapter.

CHAPTER IV.

§ 1. *Parts which do not cause referred pain.*

Throughout this paper I have neglected all such headaches as were unaccompanied by superficial tenderness. I have also pointed out, when considering the pain produced by diseases of each organ of the head and neck, that certain of the pains so produced were local and not referred. It is instructive to collect these local pains into a group and to consider their causes.

Firstly, there are certain parts which do not cause referred pain because they belong to the surface lining of the body. Such parts are the conjunctiva, the epithelium on the anterior layer of the cornea and the external auditory meatus. Such structures are in reality parts of the skin, and, when injured, no more cause referred pain than the skin of the body. The external auditory meatus not only does not cause referred pain, but is actually a part on to which pain is referred (the ear maximum of the hyoid area).

A second group of structures, which tend to cause local and not referred pain, accompanied by local and deep tenderness, is formed by the external lining of the organs of the head. Thus the periodontal membrane of the teeth does not usually cause referred, but local pain and tenderness (p. 409). The dura mater in the same way, when diseased at one spot, causes local pain over that spot associated with deep tenderness (p. 430).

Thus, here again in the head as in the body, we find that disease of the parietal lining membrane, whether external or internal, does not as a rule cause referred, but local pain accompanied by deep tenderness.

§ 2. *Megrim.*

I now reach a portion of my subject which will, I fear, run counter to the teaching of many. For I have placed megrim amongst those pains in the head which are not referred and which bear no relation to disease of organs of the head.

Of late the word megrim has been used in an exceedingly loose manner to denote almost any form of headache that is associated with vomiting.

But the megrim that has been so brilliantly described by eminent scientists[1] from observations on themselves, is a perfectly definite disturbance, differing in its course and symptoms from almost every other form of headache. The introduction of eye headaches, neuralgias, headaches referred from the stomach, and their like, into the same group as megrim has only confused and obscured one of the most definite groups of symptoms in medicine.

---

[1] Airy, Du Bois Reymond and others.

I propose, therefore, to use the word "megrim" solely for a headache presenting the following characteristics. It occurs in those engaged in intellectual work.[1] It frequently begins with a profound visual disturbance such as scotoma, hemiopia or the "fortification figure." This is followed by an intense, more or less localised, headache, which slowly reaches a certain intensity and then frequently culminates in violent vomiting.

Now, this group of symptoms is so characteristic and has been so well observed that the text-books of medicine contain practically all that is to be said about the course and surroundings of this affection.

But most authorities look upon some of the headaches they class with megrim, as due to some affection of the eyes, teeth, &c.

Now, I have pointed out throughout this paper, that a referred headache is caused by irritation of some organ, and this irritation is transmitted through the central nervous system. Thus, such a headache must follow a distribution that is central and not peripheral. Secondly, it is frequently accompanied by areas of superficial tenderness, such as I have described in chap. i.

Now, a careful examination of any patient suffering from true megrim, at once shows us that the character and distribution of the pain in this disorder bears no relation to that of referred pain. The tenderness in megrim has neither the character nor the distribution of that which accompanies referred pain. I can best illustrate this by the description of a case where I repeatedly had the opportunity of examining the patient during typical attacks of megrim.

Case 55.—Dr. G. W., aged 26.

March 1. At 10.30 a.m. the headache began with an intense boring pain around the left eye and over the forehead. This pain slowly shifted until it occupied a position four and a-half

---

[1] I have not yet seen a true case of megrim amongst hospital patients. All my patients were medical men.

EXPLANATION OF PLATES I. AND II.

On these plates an attempt has been made to represent the approximate limit of the areas on the head and neck which form the subject of this paper.

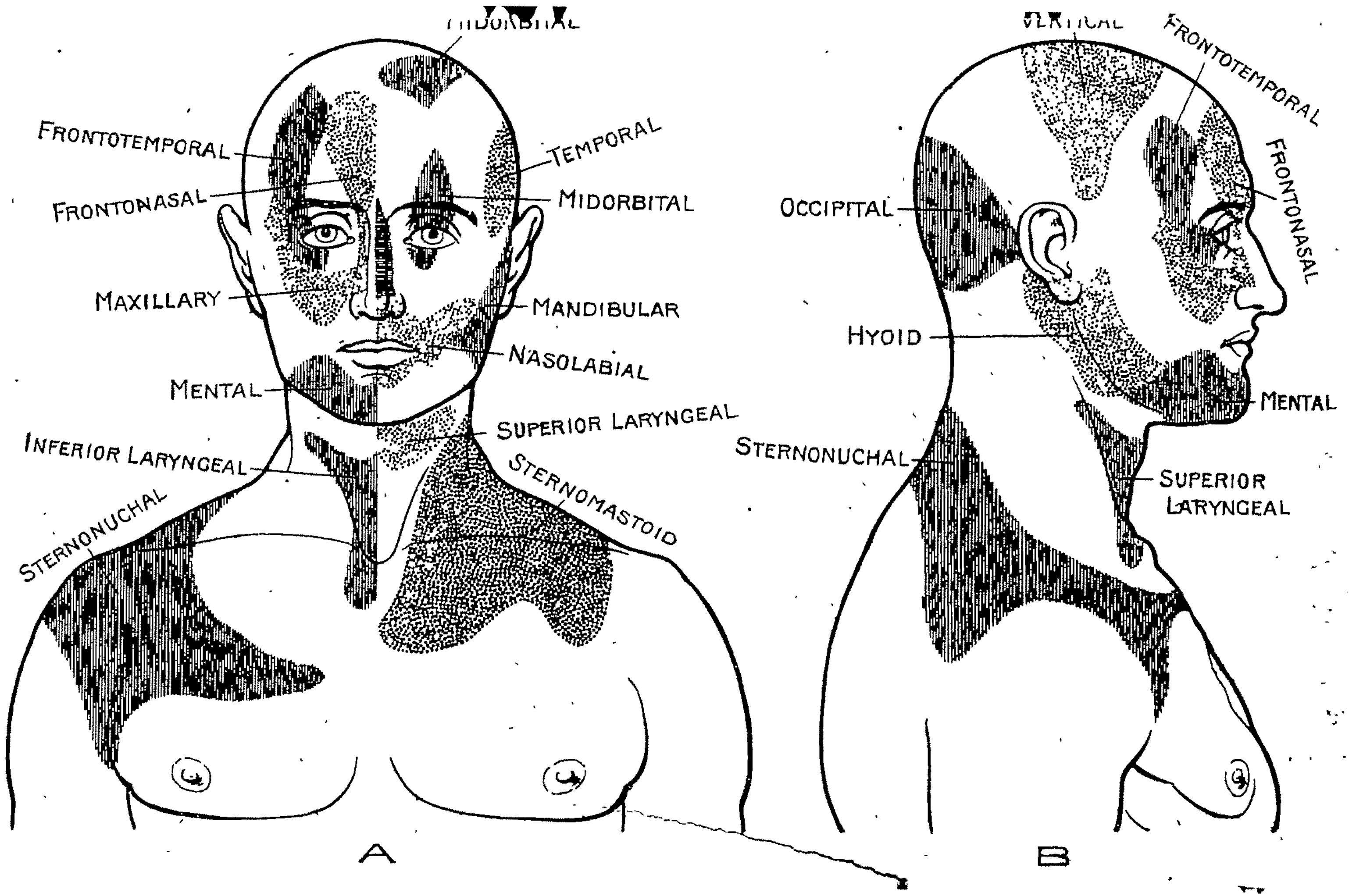
It will be noticed that the areas on Plates I. and II. are filled in with three colours—blue, red, and black. The group coloured blue consists of the occipital, parietal, vertical, temporal, fronto-temporal, mid-orbital, fronto-nasal, and the doubtful rostral areas. It is this group which is associated with areas of tenderness on the chest and abdomen (chap. iii.).

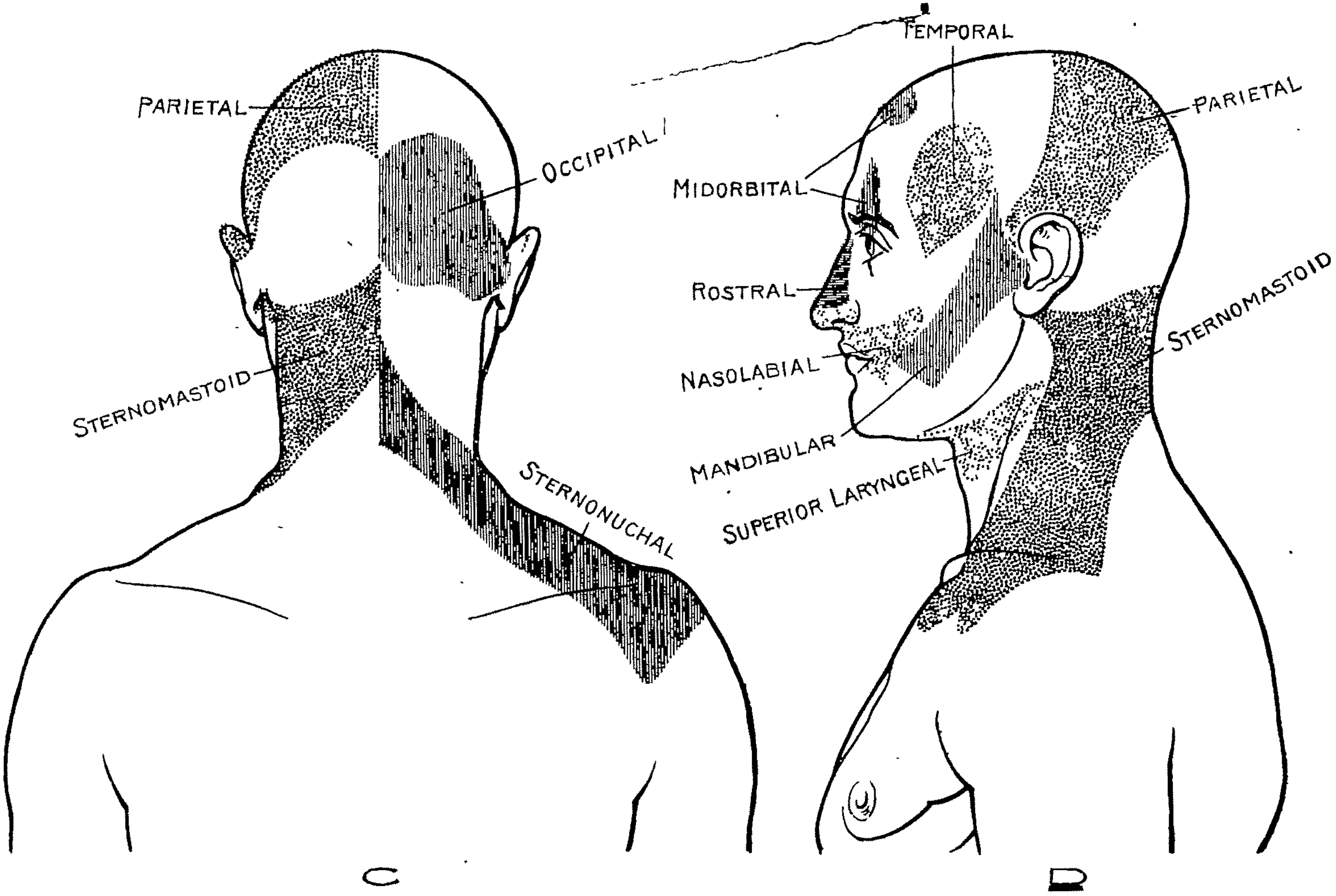
The areas coloured red become tender in affections of the organs of the head and neck, but, unlike the blue group, are not correlated with areas of tenderness on the thorax and abdomen.

The two black areas are the sterno-mastoid (third cervical segment) and sterno-nuchal (fourth cervical segment). They do not belong to the areas of the head, but are in reality the highest areas of the trunk. Thus they are not primarily affected in disturbances of any organs of the head and neck, and are not directly correlated, like the blue areas, with areas on the thorax and abdomen. On the contrary, they behave like areas on the thorax, and are associated with certain dorsal areas of the scalp (p. 457).

On Plates I. and II. I have given the full areas only; but, as I have repeatedly pointed out, a knowledge of the so-called "maxima" is of much greater practical importance than that of the complete areas. For clinical purposes the presence of a single maximum point shows that the area to which it belongs is affected, and the implication of the whole area does not materially add to the possibilities of diagnosis. If the full area to which the maximum point belonged were unknown, the diagnostic value of the maximum points would be much diminished. But knowing the area to which any maximum belongs, the presence of this maximum only is required for diagnosis. I have, therefore, given fig. 61 to shew the maximum points of those areas shewn in full on Plates I. and II.

My best thanks are due to Mr. W. J. Griffiths for the energy and ability expended over the drawings and plates in this paper.


FRONTOTEMPORAL
FRONTONASAL
MAXILLARY
MENTAL
INFERIOR LARYNGEAL
STERNONUCHAL
TEMPORAL
MIDORBITAL
MANDIBULAR
NASOLABIAL
SUPERIOR LARYNGEAL
STERNOMASTOID
A
FRONTOTEMPORAL
FRONTONASAL
OCCIPITAL
HYOID
MENTAL
STERNONUCHAL
SUPERIOR LARYNGEAL
B


PARIETAL
OCCIPITAL
STERNOMASTOID
STERNONUCHAL
C
TEMPORAL
MIDORBITAL
ROSTRAL
NASOLABIAL
MANDIBULAR
SUPERIOR LARYNGEAL
PARIETAL
STERNOMASTOID
D

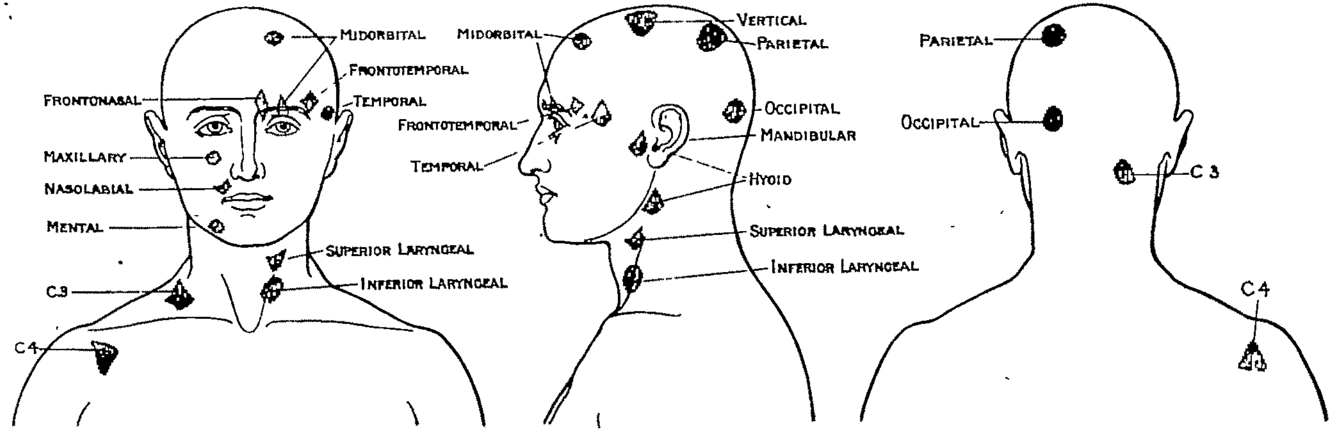


Fig. 61.

To Show the Maximum Points of the Areas drawn in full on Plates I. & II.

inches above the zygoma and one inch from the middle line of the scalp. This patch was about an inch to an inch and a-half in diameter, and lay just in front of a vertical line from ear to ear on the left side of the head. After the headache had shifted to this position he still had an intense boring pain in the left eye. This attack was not preceded by visual phenomena, which only rarely occur in his attacks. The headache continued till 4.30 p.m., when he vomited. After this he slept, and woke in the evening free from pain and discomfort.

During the attack he was kind enough to allow me to examine his sensation thoroughly. He did not flinch when the hair was gently pulled or the skin picked up either over the area of pain or over any other area of the scalp. The blunt head of a pin felt blunt to him everywhere. There was no hyperæsthesia to heat or cold.

On the other hand, even the gentlest percussion was intensely painful over the area within which he complained of pain. In this way an area of deep tenderness could be percussed out having the limits I mentioned above.

His attitude was very characteristic. He half reclined in a long armchair with his head fixed by the cushion and his arm and his body jambed between the arms of the chair, so that all shaking and movement was impossible. If I held his head between my hands and shook it gently, it caused him agony. Rising from the chair caused him great pain, and going upstairs was an impossibility. Thus he did not resent any manipulations that did not press upon the tender place or shake the skull in any way.

He has very slight hypermetropia (+ 0·75.D.) which has been corrected without the least effect upon the frequency or severity of his attacks.

He is always constipated before the attack. His urine is normal.

Pulse not altered in rate or tension.

Now compare with this an attack of "sick headache," in which referred pain is the main factor. The patient has pain over the temples, and sits or lies with his head tightly clasped between his hands. In some cases he ties a bandage around his head, or applies a cold compress to the tender spot to ease the pain. Shaking his head does not appreciably increase the headache. He can rise and go upstairs. But he resents the least pulling of the hair or picking up of the skin over the painful spot. Thus he is found to have an area of superficial instead of deep tenderness, and this area will be found to correspond to one or more of the areas I have described in chap. i.

Sufferers from true megrim are of such a high order of intelligence that their own observations are very valuable. I will now give the observations made for me by Dr. F. on himself. The attack began on getting up at 9 a.m. on October 17. The vision became double, and then a central scotoma developed, which grew bigger and bigger. The diminution in vision became so great that he could not recognise people who entered the room. Twenty minutes after this the headache began as an intense boring pain over the inner part of the right eyebrow. It radiated over the right side of the forehead, and occasionally passed over a little to the left of the middle line. Whenever he moved there was an intense pain over the vertex to the right of the middle line. At no time was there any superficial tenderness of the scalp. He could not move his head, laugh, speak, or go upstairs on account of the pain these actions produced. About half an hour after the headache had begun, vomiting came on, associated with nausea. It was excruciatingly painful. This lasted till the afternoon, when he slept.

Those who suffer from true megrim are certain that they suffer from no superficial tenderness of the scalp during their attacks. The late Dr. James Anderson was kind enough to interest himself in this investigation, and repeatedly made observations, for my benefit, on the megrim attacks to which he was a martyr. He assured me that after repeated trials he was unable to find any superficial tenderness in himself during the attacks. The least percussion, however, caused him intense pain. He was hypermetropic, and had been corrected by one of our best eye surgeons without in the least diminishing the frequency or severity of his attacks.

Thus, in conclusion, I think we may say that true megrim is an affection *sui generis*, and not in any way related to referred pain.

The differences between true megrim and a referred headache may be tabulated as follows.

MEGRIM.	REFERRED HEADACHE.
1. The pain follows areas differing fundamentally in distribution from those of referred pain.	1. The distribution of the pain follows the lines laid down in chap. i.

2. The pain is associated with intense deep tenderness.	2. The pain is accompanied by more or less intense superficial tenderness.
3. Movement of the head, or anything that produces it (laughing, going upstairs, &c.) causes an increase of the pain.	3. Pressure relieves the pain, and the patient can go upstairs without producing much increase in the headache.
4. The headache may be preceded by visual phenomena of the highest order, such as "fortification figures" scotoma, &c.	4. The headache is never preceded by these highly developed visual phenomena, but may be accompanied by amblyopia or coloured flashes of light.

CHAPTER V.

Do these Areas correspond to the supply of Peripheral Nerves?

The course of a nerve may be mapped out in three ways :—

(1) Anatomically—The nerve may be traced to its furthest point by dissection.

(2) The nerve may be divided and the anæsthetic area, so produced, mapped out. This method shows solely what part of the skin is supplied by this nerve alone. For should any portion of the skin be supplied by two nerves, division of one nerve will not render that part of the skin anæsthetic.

(3) The method adopted by Sherrington, which may be called the method of remaining æsthesia. He divides all

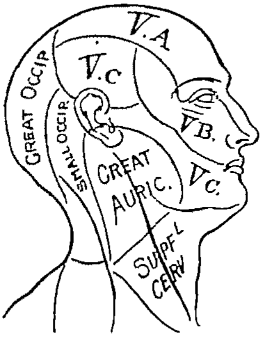


FIG. 54.

To show the distribution of the main branches of the fifth nerve and cervical plexus, determined by dissection. (For this figure I am indebted to Professor Thane.)

the roots above and below one single root, and thus leaves a sentient field in the centre of a desert of anæsthesia.

Let us see how far these methods enable us to trace the supply of the main nerve trunks of the head and neck.

The anatomist describes the supply of the branches of the fifth nerve somewhat as is represented on fig. 54. The ophthalmic division supplies the greater part of the nose and conjunctiva, the upper lid, and passes up to supply the hairy scalp as far back as the parietal eminence. The second division of the fifth nerve supplies the upper lip and cheek, whilst the third division supplies skin over the chin, part of the lower jaw, the greater part of the ear, and a portion of the hairy scalp over the temporal fossa.

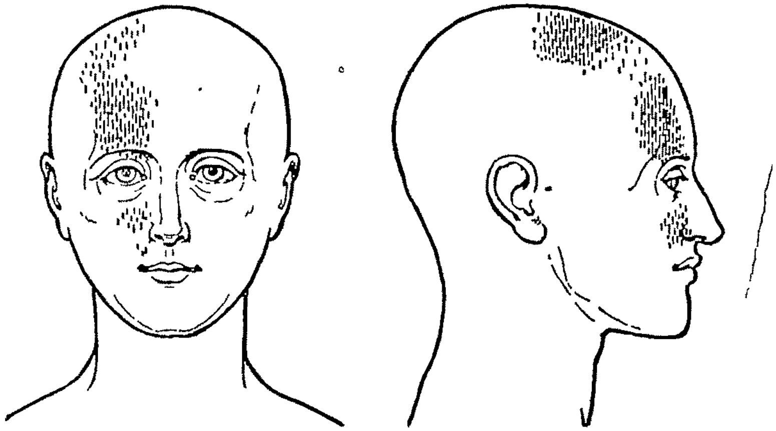


FIG. 55.

To show the loss to pain, heat and cold produced by excision of all the cutaneous branches of the ophthalmic division, and the whole of infra-orbital (second division) of the fifth nerve on the right side. The loss to touch was still less marked than the loss to other forms of stimulation.

A line drawn along the posterior border of the black area on this figure, will represent the anterior border of the area supplied by the third division of the fifth nerve (by Sherrington's method of "remaining anæsthesia.")

When the supra-orbital, supra-trochlear, and infra-orbital portions of the fifth nerve are destroyed before they reach the face the resulting anæsthesia is much less extensive than we should expect from the anatomist's description of the supply of these nerves. Fig. 55 shows the extent of the analgesia or loss to pain caused by division of these nerves for the relief of facial neuralgia. The loss to heat and cold closely followed the distribution of the loss to painful stimuli, but the loss to touch was less extensive.

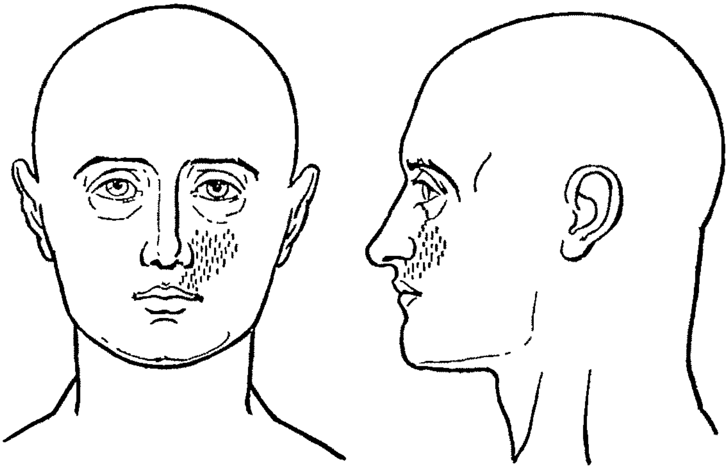


FIG. 56.

To show the loss to pain, heat and cold, produced by excision of the whole of the infra-orbital (second division) of the fifth nerve There was no absolute anæsthesia to touch, but only a diminution in sensibility over a small area on the left cheek.

In another case Mr. Horsley divided the infra-orbital division only by following it back to the posterior wall of the antrum (fig. 56). The loss to pain stimuli consisted of an exceedingly small patch over which sensation was diminished, and there was practically no anæsthesia to touch.

I give a third figure (fig. 57) to show the result produced by dividing the third division of the fifth nerve at the foramen ovale (Salzer's operation). Here the resulting analgesia or loss to pain stimuli corresponds more nearly to the anatomist's description of the supply of this nerve. There was no absolute loss to touch anywhere (excepting within the area of the operation scar). But a touch was said to feel "wooden." Heat and cold followed the distribution of the analgesia.

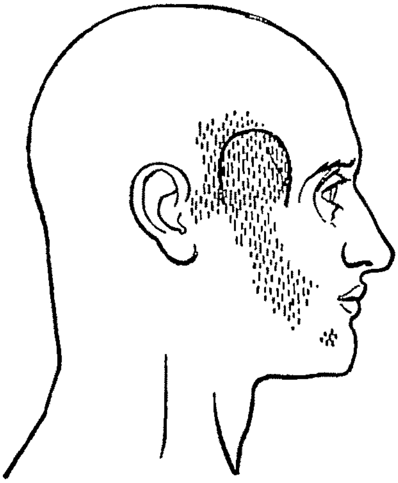


FIG. 57.

To show the loss to pain, heat and cold, produced by excision of the whole of the third division of the fifth nerve (Salzer's operation).

The curved black line represents the skin incision. Within this area the loss is more profound owing to the division of anastomosing nerves.

Thus, in all cases where one or more branches of the nerve are divided the loss to pain stimuli falls far short of the anatomical supply of the nerve, and the loss to touch stimuli is ridiculously small considering the gravity of the lesion.

If, however, all three branches are cut (fig. 58), a change takes place, and both the analgesia and anæsthesia are of considerably greater extent than when any two branches only are divided (compare fig. 58 and fig. 55).

Thus, both the anatomical and the destructive methods have grave disadvantages. The former cannot tell us the limits of the functional activity of the nerve; the latter only gives us the minimal area supplied by any branch.

The only satisfactory method of mapping out the supply of these branches is that adopted by Sherrington. He has been kind enough to allow me to include figs. 59 and 60 obtained by him from observations on monkeys, which are as yet unpublished. In order to map out the distribution of the ophthalmic division of the fifth he cuts the posterior roots of the fourth, third and second cervical nerves, together with the second and third divisions of the fifth on one side. Eight hours or more afterwards he finds he can obtain a reflex by pinching the skin over the area in fig. 59. Thus, this area corresponds to the whole area supplied by the ophthalmic division of the fifth, for all the nerves below have been divided. In the same way he traces the posterior border of the fifth nerve by dividing all the upper cervical posterior roots. A glance at fig. 59 shows how the area of sensation which remains oversteps the limits described by the anatomist, or those of the analgesia produced by dividing the nerve itself.

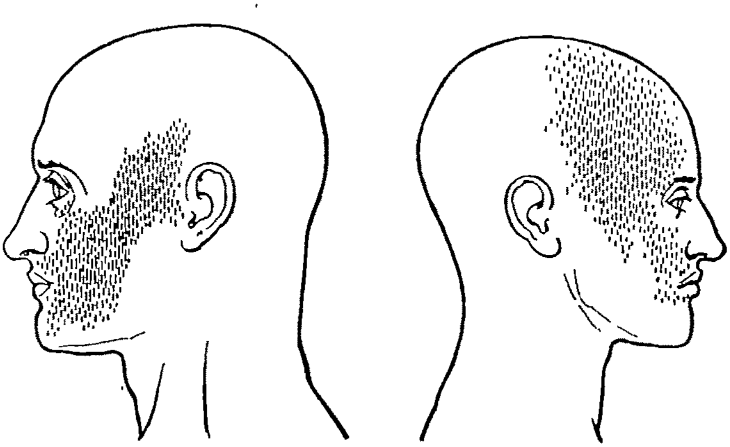


FIG. 58.

The *left hand* figure shows the loss to pain, heat and cold, produced by dividing the second and third divisions of the fifth nerve.

A line drawn along the upper border of the shaded patch represents the lower border of the supply of the ophthalmic division of the fifth nerve by the method of "remaining æsthesia" (Sherrington). Compare Sherrington's observation on the monkey (fig. 59).

The *right hand* figure represents the loss to pain, heat and cold, produced by destruction of all the cutaneous branches of the fifth nerve.

A line drawn along the posterior border of the shaded patch represents the anterior border of the supply of the posterior root of the second cervical nerve by the method of "remaining æsthesia." Compare Sherrington's observations on the supply of this root in the monkey (fig. 60).

Fig. 59 and fig. 60, which I owe to Dr. Sherrington's kindness, also show the upper and lower limits of the second, third and fourth cervical roots observed by his method.

Clinically we possess the means of mapping out the distribution of at any rate some of the branches of the fifth nerve by practically the same method as that adopted by Sherrington. For after dividing the middle and lower divisions of the fifth nerve for facial neuralgia, a sentient area is left which represents the supply of the uninjured ophthalmic division of this nerve. A comparison of fig. 58 (left figure) with fig. 59 (right figure) shows how closely this area in man resembles that found by Sherrington in

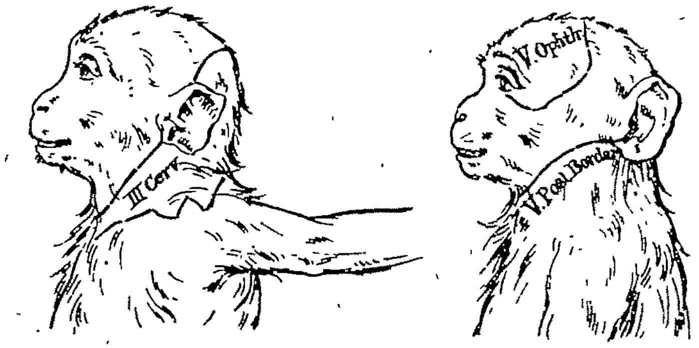


Fig. 59.

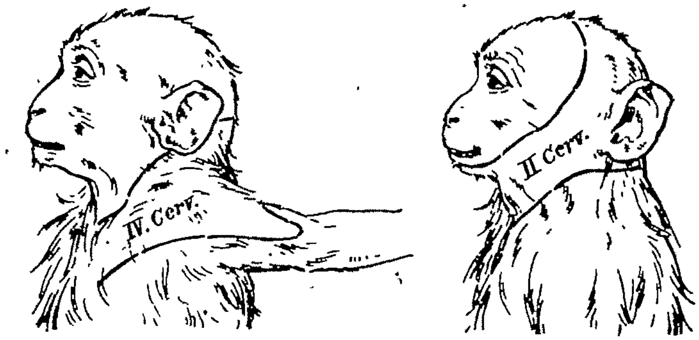


Fig. 60.

To show the areas supplied by the ophthalmic division of the fifth, by the whole fifth nerve, by the second, third and fourth cervical nerves in the monkey, determined by Sherrington's method.

Compare the right hand drawing of fig. 59 with the left hand drawing of fig. 58, and the anterior border of the second cervical on fig. 60, with the posterior border of the anæsthetic area on the right hand drawing of fig. 58.

the monkey. In the same way the area which remains sensitive after division of all three branches of the fifth nerve (fig. 58) represents the anterior border of the posterior root of the second cervical.

Now a comparison of the areas in chapter i. with the areas of distribution of the cervical nerves and of the branches of the fifth shows a difference and at the same time a likeness. Thus, Sherrington's fourth cervical bears a resemblance to my sterno-nuchal area, and his third resembles my sterno-mastoid area.

But although there is a likeness the differences are profound. The overlap in the supply of these peripheral branches is enormous. Thus, the anterior border of the supply of the posterior root of the third cervical touches the posterior border of the third division of the trigeminal.

Thus, the whole large area of the second cervical is superposed on skin already supplied by the trigeminal in front and the third cervical behind.

Thus, I think we are forced to conclude that my areas of tenderness represent some more central nervous arrangement than the posterior roots of the cervical plexus and main branches of the fifth nerve.

CHAPTER VI.

Theoretical.

We have now seen that, although the boundaries and form of the tender areas are foreshadowed in the distribution of the peripheral branches of the nerves of the head, these areas represent neither the posterior roots of the cervical nerves, nor the three branches of the trigeminal. In Part i. of this work I came to a similar conclusion with regard to the significance of the tender areas on the body. I then tried to show that although they did not represent the supply of posterior roots, they corresponded to the distribution of segments of the spinal cord. One might then suppose that the analogous areas on the head and neck also represented cephalic segments. But so much shifting has taken place in the head that we cannot say whether each area on the head is be considered the equivalent of a whole zone from dorsum to venter on the body or only to a portion of such a zone. Thus we are unable to say whether the temporal area on the scalp represents the supply of a segment in the cephalic nervous system equivalent to the seventh dorsal area on the body; or whether the temporal maxillary and naso-labial must be combined to produce the homologue of a complete sensory band on the body.

There are, however, certain points in which these areas on the face and scalp seem to retain the marks of the changes which occurred in the development of the face and head in the human embryo.

The face is developed by the union of three tissue masses. The mesial nasal process descends from above, and finally becomes the bridge of the nose (on the surface).[1] On each side of this mesial portion the lateral nasal process descends between the mesial process and the eye to form the ala and side of the nose. It also possibly enters into the formation of the mesial structures of the upper jaw. The maxillary processes come inwards on each side to meet in the middle line, and form the upper lip.[2]

Now, the nasal portion of the fronto-nasal sensory area bears upon it the traces of some such downward growth, whilst the maxillary area shows similar traces of a growth inwards from the sides of the face.

The tongue is developed in two portions. The front of the tongue is formed from a tuberculum impar in connection with the ventral portion of the mandibular arch. The posterior and postero-lateral portions are formed from a mass produced by the coalescence of the ventral portions of the hyoid and of the third postoral arches. Thus the mandibular hyoid and third postoral arches all take part in the formation of the tongue.

---

[1] His, "Anatomie Menschlicher Embryonen," Leipzig, 1885 (a large number of His's figures are reproduced in "Quain's Anatomy," tenth edition, vol i., part i.).

[2] *Cf.* Hochstetter, *Verhandlung d. Anatomischer Gesellschaft*, 1891, p. 145, and 1892, p 181.

Now, the sensory supply of the tongue stands in relation with the same nerve elements that supply three separate areas on the face. The anterior part of the tongue stands in relation with the mental area on the face. This area lies over the front of the lower jaw and is connected with the incisors, canine and first bicuspid on each side. The lateral part of the tongue is in connection with the hyoid area—an area which is closely connected with the tonsil and membrana tympani. The posterior part of the tongue stands in relation with the superior laryngeal area of the neck; and this area is in close connection with the upper part of the larynx. Thus an area associated with structures developed from the mandibular arch supplies the front of the tongue; an area connected with structures developed from the hyoid arch supplies the sides of the tongue; and an area in connection with structures developed from the third postoral arch stands in relation with the back of the tongue.

The next area below, the inferior laryngeal, supplies the larynx proper, which is developed from the fourth postoral arch. It is interesting to notice that the sensory nerve through which this reference from the larynx to the inferior laryngeal zone on the skin must take place, is the recurrent laryngeal. This nerve curls round the arch of the aorta, which we know to be the representative of the vessel of the fourth postoral arch.

The tragus is developed from the mandibular arch, the lobule and antitragus from the hyoid arch.

Now, what I have called the mandibular sensory area supplies the tragus, whilst the lobule and antitragus are supplied by the hyoid sensory area.

During the development of the neck the somatic structures from behind come forwards to enclose the branchial structures in front, which belong to the head. His states that the anterior border of the sterno-mastoid muscle fairly represents the point at which the somatic structures meet the structures of the anterior part of the neck (of branchial origin).

Now the same line fairly represents the anterior border of my third cervical area, and we saw reason to believe

(p. 457) that the third and fourth cervical areas of the neck behaved like areas of the body, and not like areas of the head and neck. For they are associated with frontal tenderness on the scalp, and thus behave differently from any other areas of the neck or face.

Thus these tender areas of the face and neck seem to retain the marks of the developmental processes which have led to the formation of the human head. Further than this I do not think we can justifiably go at present.

In chapter iii. I attempted to show that organs of the thorax and abdomen not only refer on to areas of the body, but on to areas of the head.

Now these areas on the body represent the supply of segments of the spinal cord. Thus we are tempted to conclude that the areas on the scalp which stand in correlation with them represent a similar segmentation of the cephalic nervous system.

These areas which stand in relation to the areas of the body lie above the following line.—It starts from the side of the nose, runs upwards, and along the lower border of the bony orbit. Thence it runs with a sloping course upwards to a point above the ear. It then dips sharply downwards to end below the occipital protuberance.

There seems to be a fundamental difference between the areas which lie on the dorsal side of this line to those which lie below it. I cannot help thinking that if there are any traces of segmentation in the cephalic nervous system akin to that in the spinal cord, we must seek for it amongst these dorsal areas of the scalp and not amongst those of the face or neck.

Why should disturbances of the thoracic and abdominal viscera cause pain and tenderness within the distribution of the trigeminal?

On examining the supply of the dorsal areas of the scalp from peripheral nerves we find that they receive branches partly from the fifth nerve, and partly from the posterior roots of the second, and possibly third, cervical nerves. The great bulbar nucleus (so-called ascending nucleus) of the trigeminal descends as low as the second cervical, and is probably a continuation above of the nuclei of the posterior roots of the cervical nerves.

Thus, if we take the sensory nucleus of the trigeminal and add to it the nucleus of the posterior root of the second cervical, the sum total must consist of at least eight primitive nuclei akin to the nuclei of posterior roots. Of these one belongs to the second cervical, one to the first cervical, which no longer has connection with a single peripheral root, but is partly in fifth and partly in the second cervical nerve bundles. The remaining six nuclei, corresponding to the rostral, fronto-nasal, mid-orbital, fronto-temporal, temporal and vertical areas, belong to the head.

Originally each of these hypothetical nuclei received a direct ramus which passed into it at its own level. But for some reason we do not understand, these rami disappeared and the fibres now reach their nuclei by an indirect route, through the great trunk of the fifth nerve. Hence the enormous downward prolongation of the bulbar (so-called ascending) root of the fifth.

All the thoracic and abdominal viscera which refer into the dorsal areas of the scalp, are supplied by the nerve which must be called the vago-glosso-pharyngeal. Now Gaskell has pointed out that the vagus and glosso-pharyngeal represent the visceral branches of a set of nerves, whose somatic sensory roots are to be found in the sensory portion of the fifth. If this is true it is not surprising that the impulses passing up the vagus should be referred to the area of the fifth. Why, however, they should stand in relation to the areas supplied by segments of the spinal cord remains inexplicable.

Speculation on such points is easy but liable to be unprofitable, and I must crave my readers' indulgence for the extent to which I have entered into possibly fanciful theories in this chapter.

It is now my pleasant duty to thank those who have helped me in various ways during this investigation.

My thanks are due to the medical and surgical staff of University College Hospital for the kind way they have permitted me to use their cases.

To the staff of the City of London Hospital for Diseases of the Chest (Victoria Park) I return my thanks for the exceedingly liberal way in which they have allowed me to make use of the cases that came under my charge when house physician. To the Lay Committee of this Hospital I also return thanks for extending my period of office beyond the usual term.

I beg also to thank the medical and surgical staff of the National Hospital for the Paralysed and Epileptic (Queen Square), for permission to use my observations on their cases.

My especial thanks are due to the staff of the National Dental Hospital, for the exceedingly liberal way they have permitted me to use their cases without let or hindrance.

I beg to offer my especial thanks to the following gentlemen:—To Mr. Horsley, for permission to study his cases of trigeminal neuralgia. To my late chief, Dr. Buzzard, for his liberality and kindness to me. To Mr. Sidney Spokes, for much sympathy and help in the dental section. To Mr. Tweedy and Mr. Marcus Gunn for their kindness during the time I acted as their assistant. To my friend, Dr. Sherrington, for much help and for permission to embody the results of some of his unpublished work, in this paper. And, lastly, to my colleagues, the residents at the Victoria Park Chest Hospital, my thanks are due for much kind thought and valuable aid in diagnosis.

LOCAL ANÆSTHESIA AS A GUIDE IN THE DIAGNOSIS OF LESIONS OF THE UPPER PORTION OF THE SPINAL CORD.

BY M. ALLEN STARR, M.D., PH.D.

Professor of Diseases of the Mind and Nervous System, College of Physicians and Surgeons, New York.

THE importance of the careful analysis of special symptoms and of their relation to localised lesions in the nervous system can hardly be exaggerated. There is perhaps no better proof of this statement to be cited than the exact results which have been reached by the study of paralysis and anæsthesia following transverse lesions in the spinal cord. It has been possible to determine the various levels or segments of the cord which are in functional relation with certain muscles and regions of the skin. This has been most exactly worked out for the lower half of the body and its relation to the lower portion of the spinal cord by the independent researches of Thorburn[1] and myself,[2] and recently by Head.[3]

---

[1] The " Surgery of the Spinal Cord," 1889 ; also BRAIN, 1893, p. 373.

Thorburn's criticism that my diagram shows no skin areas connected with the fourth lumbar and second sacral nerves is not quite just, as the following quotation from my article shows :—

" The *fourth zone* is of a similar shape to the third, but more extensive, a greater surface on the back of the thighs being involved, and the anæsthesia extends in a band almost as low as the popliteal space. This area has been established clinically in several cases : there is as yet no autopsy to determine its lesion, but since the smaller zone is due to lesion at the third sacral segment, and the next larger zone is due to lesion in the fifth lumbar segment, it is allowable to conclude that this region corresponds with the second and first sacral segments. In thus outlining four zones of the skin and assigning them to various segments of the sacral portion of the spinal cord, it is not my intention to lay down artificial boundaries or to affirm that all cases will exactly coincide. I only wish to show that as the cord is invaded by disease from below upward, the area of the skin which becomes anæsthetic increases in extent, and that the shape of the area is characteristic ; so that from the study of the area the extent of the lesion can be determined. The *sixth zone*

As a further contribution to this subject I propose in the present article to consider the anæsthesia produced by lesions of the cervical segments of the cord. This will be less difficult since cases are not wanting both in the recent literature and in my own experience to aid in this study.

A few remarks regarding the anatomy of the cervical segments of the cord and the relation of these segments to the cervical vertebræ may not be out of place. It is to be remembered that there is no natural boundary between adjacent segments of the human cord. The nerve roots issue from the cervical cord in a continuous series, and it is only by following out individual nerve roots to the various points of exit, through the dura mater, that it is possible to trace any one particular root into the cervical nerve to which it belongs. If, on the other hand, any nerve be traced into the cord, it will be found to sub-divide, some portions passing directly into the gray matter, others turning downward and others ascending in the cord, so that any one root really reaches several levels of the cord.

It cannot therefore be affirmed that individual segments of the cord are in any special or direct relation with single cervical nerves, and therefore the symptomatology of spinal cord lesions in the cervical region at different levels will be different from the symptomatology of lesions of the individual cervical nerves, or even of lesions of the nerve roots.

---

of anæsthesia is produced by a lesion of the third lumbar segment. When the third lumbar segment is diseased, the entire back of the thighs and legs is anæsthetic, and the front of thighs is also anæsthetic, except over a funnel-shaped zone which extends from above downward, the narrow tube of the funnel reaching along the shin even to the foot. This zone will probably be separated later into two separate parts corresponding to lesions of the fourth and third lumbar segments. There is not as yet a sufficient number of cases to warrant such a distinction. The exact limits of anæsthesia on the feet are still uncertain, and no more exact statement than that given is warranted."

It seemed to me better to abide closely by actual facts than to assert conclusions without the pathological basis of exact spinal lesions. I do not think any definite conclusions can be deduced from cases of pressure on the cauda equina. While I think it quite probable that Head and Thorburn are correct in assigning the sole and outer side of the foot to the first sacral segment, I know of no case of *spinal cord lesion* with autopsy to establish this fact.

[2] *American Journal of Medical Sciences*, 1892, and *American Journal of Neurology*, 1884

[3] BRAIN, 1893, p. 44.

In discussing lesions of the cervical enlargement of the cord, authors have usually been contented to describe the symptoms produced by destruction of the lower, middle and upper thirds of the enlargement, and this is perhaps sufficiently accurate for all purposes of diagnosis. Nevertheless, after the study of the cases here collected I shall attempt to assign to each segment its special function, although it is admitted at the outset, that scientific precision rather than practical use will be the chief result.

The relation of the cervical segments of the cord to the spines and bodies of the vertebræ is by no means a constant one. It will be remembered that Gowers in 1884 published a diagram to demonstrate these relations which has been accepted ever since that time. Gowers says:[1] "The first three cervical spines are opposite the origin of the third, fourth and fifth cervical nerves. The sixth and seventh pairs arise opposite the intervals between the fourth and fifth, and the fifth and sixth, cervical spines respectively. The sixth cervical spine corresponds to the origin of the eighth cervical nerve, and the seventh cervical spine to the first dorsal nerve. Thus the cervical enlargement of the cord which ends at the origin of the first dorsal nerve corresponds nearly to the bodies and spines of the cervical vertebra."

While this summary is undoubtedly very nearly correct it must be admitted that it does not hold for all cases. In 1891, Kraus called attention[2] to the fact that fractures of the sixth and seventh cervical vertebræ crushing the cord often produce very different symptoms in different individuals, the extent of the paralysis being by no means uniform, although the site of the fracture is the same. This fact was confirmed in 1892 by the series of cases published by Church and Eisendrath.[3] The facts presented by these authors can only be explained upon the supposition that in different individuals the relation of the segments to the vertebræ differs.

Quain in the last edition of his "Anatomy" admits this fact. Quain states that the lower limit of the cervical enlargement

---

[1] "Diseases of the Spinal Cord," 1884, p. 6.

[2] *Zeitschrift für Klin. Med.*, xviii.

[3] *Amer. Jour. Med. Sciences*, April, 1892.

lies opposite the body of the first or second thoracic vertebra. But inasmuch as the thickness of this body is at least an inch, it is evident that this implies a decided variation in the relation of individual segments of the cord to the vertebræ. This variation is also brought out by a diagram of Reid[1] shown in fig. 1. The position of attachment of the nerve roots to the spinal cord in six adult subjects and the relation of these to the spines of the vertebræ are shown in the diagram. It is evident from its study that no exact conclusion can be drawn as to which segment of the cord is destroyed in a fracture of one vertebra. The possible result, for example, of a fracture of the seventh cervical vertebra would be a destruction of either the eighth cervical segment or the third dorsal segment or segments between these in different cases. It is evident, therefore, that while Gowers' statement may be taken as representing the average

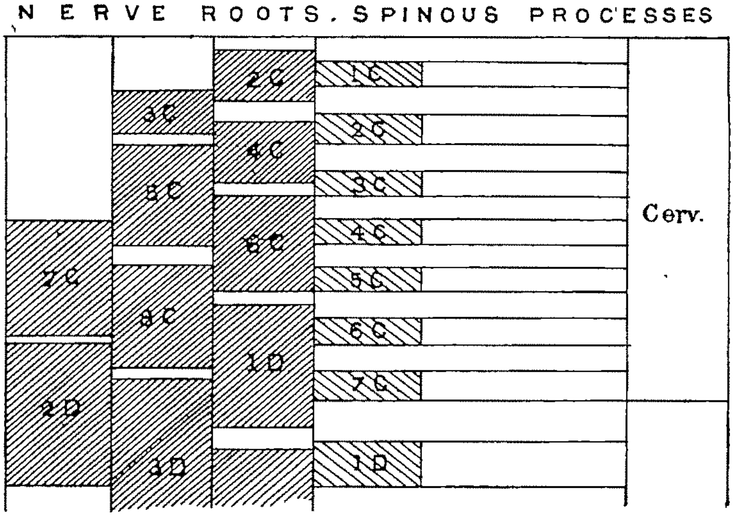


FIG. 1.

Chart showing the limits of the variations in the topographical relation between the superficial origins of the nerve roots from the spinal cord and the spinous processes of the vertebræ.—Reid.[2]

---

[1] *Jour. of Anat. and Phys.*, xxiii., 1889.

[2] *Jour. of Anat. and Phys.*, 1889, vol. xxiii., p. 341.

condition, there are considerable variations in the relation between the cord and the spine.

There is another fact which must be stated as a necessary preliminary to the study of anæsthesia due to spinal lesions, the fact which has recently been brought out by the investigations of Sherrington.[1]

Sherrington found, in an attempt to determine the area of the body functionally related to the various posterior nerve roots in the monkey, that division of one root alone was not attended by any anæsthesia, and that to determine the area of skin supplied by a single dorsal nerve it was necessary to divide at least three dorsal nerve roots.[2]

The conclusion to be drawn from Sherrington's researches, as shown in his diagram (fig. 2), is that every part of the skin is supplied with sensation by three sensory nerve roots, whose peripheral branches overlap one another or anastomose. In order to deprive the area of skin of sensibility, all three nerve roots must be cut. This fact gives the rational explanation to the correlated fact which has been observed by clinical investigators, and to which attention was called especially by Horsley and Gowers in 1889;[3] namely, that in a transverse lesion of the spinal cord, the

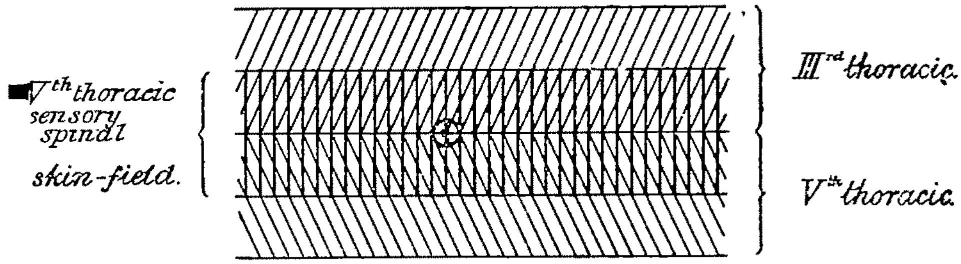


FIG. 2.

---

[1] "Experiments in examination of the Peripheral Distribution of the Fibres of the Posterior Roots of some Spinal Nerves."—*Philosoph. Transactions of the Royal Society of London*, vol. clxxxv., B. p. 641. Charles S. Sherrington.

[2] I have confirmed this observation in man; for in a case of spinal injury recently operated upon by Dr. McCosh for me, one pair of dorsal nerves was divided, but no anæsthesia was produced.

[3] *Medico-Chirurgical Transactions*, vol. lxxii., p. 21: "Report of a Case of Tumour of the Spinal Cord."

band of anæsthesia about the body was uniformly at a level from two to four inches lower than the level of the lesion in the cord. If, for example, as in my first case, the level of anæsthesia is about the body at the level of the nipples, that is, corresponding to the fifth dorsal nerve, the lesion in the cord cannot be lower than the fourth dorsal nerve or fourth dorsal segment. For were it at the fifth dorsal segment, the fourth dorsal nerve being still intact would supply with sensibility the skin in the fifth dorsal area. This fact is of practical importance in determining the position of a lesion in the cord, such as a tumour, and justifies the dictum of Horsley, that in removing tumours from the cord the tumour is to be sought from two to four inches above the level indicated by the line of anæsthesia. I can confirm this from my own observation in two cases of spinal cord tumours recently successfully removed, and which will be reported elsewhere.

This fact of collateral supply of skin by adjacent nerves will also explain the clinical fact that the distribution of anæsthesia upon the arms in lesions of the cervical cord is not always uniform.

With these preliminary observations, I will now proceed to relate a number of cases from my own experience and from reliable observers, which demonstrate the various symptoms produced by lesions at various levels of the upper dorsal and cervical segments.

Case I.—Transverse lesion at the fourth dorsal segment of the cord. Unusual symptoms. Remarks.

H. D., aged 46, had always been in good health until his present illness, though he had a chancre with slight secondary symptoms of syphilis in 1882. On December 4, 1884, without any apparent cause he began to suffer from pain in his back, and the next day he became suddenly paralysed in legs and body, lost sensation below his navel and lost control of his bladder and rectum. The condition of total paraplegia had remained up to the time I first saw him, in consultation with Dr. L. A. Sayre on October 24, 1888; and remains at present (1894). His condition on October 24, 1888, was as follows:—

He lay upon a smooth, hard bed, totally naked, and apparently in considerable pain, and was turned every ten minutes from one side to the other by his attendants, by being lifted on a sheet, as this was the least painful method. The pain caused by the dorsal position was too great to be endured. His pain was referred to the spine at the fifth dorsal vertebra. There was no deformity there, but he was extremely tender to touch, and to a hot sponge over an area three inches wide the centre of which was at the fifth dorsal spine. He was accurate in all his statements.

Sensation.—There was a condition of absolute anæsthesia to touch, temperature, pain, and muscular sense, below the level of a line extending round the body at the level of the fifth dorsal spine. This line did not follow the course of the fifth intercostal space but in front was just opposite to the level behind, being about an inch below the nipples. It was not an absolutely straight line, however, appearing to dip down a little behind on the left side. (Fig. 3.) Above this anæsthetic line there was a zone of very intense hyperæsthesia of three inches in width, its upper limit fading off into that of normal sensation. The hyperæsthesia was so extreme that any touch was painful, and hence he would not allow even the bed clothes to cover his body, and he always lay naked in a very hot room. The sensation of all parts above the hyperæsthetic area was perfect. *Whenever he was touched at any spot in the anæsthetic area he felt pain in the back at the fifth dorsal vertebra.* This statement was repeatedly confirmed. A touch or prick on the foot or abdomen was felt in the back but not at the point touched.

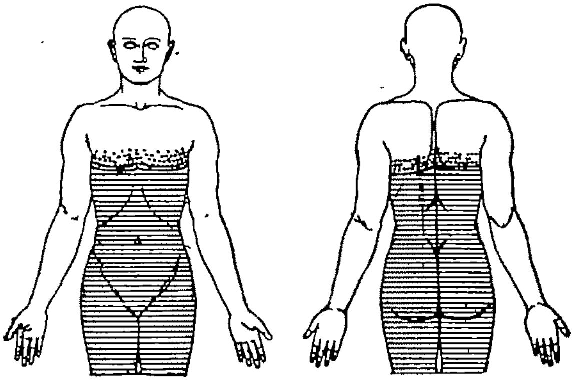


FIG. 3.

Case I.—Starr. Anæsthesia in lesion at fourth dorsal segment.

Motion was absolutely lost in all muscles of back, body, and legs below the fifth dorsal vertebra. It was perfect in the arms. The muscles were somewhat atrophied uniformly, from disuse. They were all relaxed, not at all rigid or contractured, but were abnormally sensitive to percussion and contracted on any strong sensory stimulus like that of a sharp prick on the skin over them. Their reaction to electrical stimulus both faradic and galvanic was normal, and even in the seventh intercostal space reaction was good. His sensitiveness prevented electrical tests of the sixth and fifth and fourth intercostal muscles. His feet were dropped, but not held rigid by contracture.

Reflexes.—Epigastric reflex lost. Gluteal, cremasteric and plantar reflexes normal. Abdominal and patella reflexes increased, no ankle clonus. His bladder empties itself spontaneously as soon as about three ounces of urine have collected, but there is no dribbling. For two weeks after the onset there was retention and a catheter was used, but since then active incontinence has been present. His rectum has to be evacuated by digital manipulation.

Vasomotor Condition.—If he lies more than fifteen minutes on any spot it becomes red, and will within a few hours become blue and go on to a bedsore. He has had numerous bedsores, one deep one over the sacrum, which had been healed with difficulty, and scars remain. The application of heat—as by hot bottles—or of electricity, or the mere scratch of a finger nail is soon followed by a broad zone of redness. There is always more or less redness over the bony prominences. It is to avoid bedsores that he is turned every ten minutes day and night. The growth of hair and of the nails was in no way abnormal.

Remarks.—The diagnosis of spinal hæmorrhage or of hæmorrhagic myelitis seemed quite evident in this case, and its level at the fourth dorsal segment of the cord was indicated by the level of the anæsthesia.

There were certain facts which made the case an interesting one, viz.:

First, the limits of the anæsthesia. This did not follow the line of the fifth intercostal nerve and thus describe a curve convexity down around the side of the body, but was practically horizontal to the vertical axis. This confirms the observations of Church and Eisendrath (*American Jour-*

nal of Medical Sciences, April, 1892), in five cases, and also Head's statement.

Secondly, the patient, though anæsthetic below this line, felt any touch or pain in the anæsthetic region at a spot in the back at the level of the lesion. I have observed this symptom in several cases of transverse myelitis, though I do not recall any special mention of it in the literature.

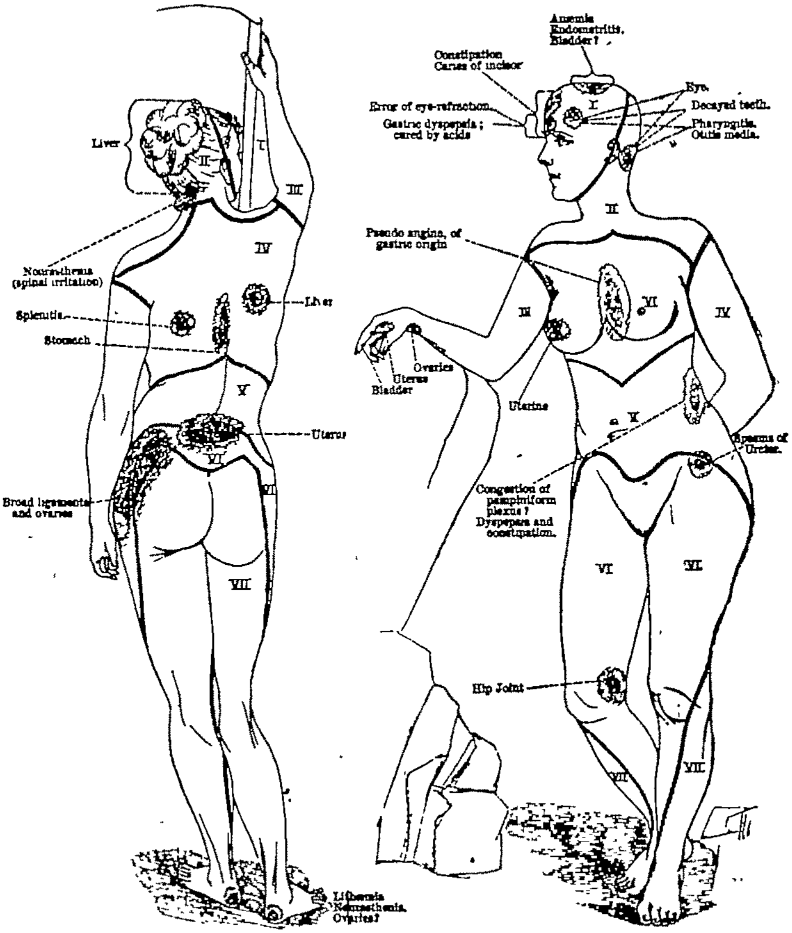


FIG. 4.

Dana. Situation of Referred Pains.

The impression sent inward from the skin evidently travels up the cord to the level of the lesion; there sets up an irritation which is then referred erroneously to the lowest area in which sensation can be felt, viz.: to the hyperæsthetic area just above the lesion. The explanation is the same given by Head in chapter vi. of his article, and long ago offered by Dana in his article on "A Clinical Study of Neuralgias and of the Origin of Reflex or Transferred Pains." *New York Medical Journal,* July 23 and 30, 1887.)[1] Such a referred sensation would not be possible in a case of total destruction of the cord, and hence like the loss of knee-jerk, may be of value in diagnosis.

Thirdly, the absence of rigidity and contraction in the legs after nearly four years of total paraplegia in spite of increased knee-jerks, is in my experience exceptional.

It is common to find a flaccid paralysis after total lesions of the cord which destroy the gray matter in its entire length, and thus remove the tonic influence of the spinal centres. It is also possible to have a flaccid paralysis in the muscles below a transverse lesion of the cord which entirely destroys a single segment, but under these circumstances the absence of knee-jerks (as first shown by Bastian) establishes the diagnosis of a destructive lesion.

In my case the knee-jerks were not lost, and the condition of sensibility just mentioned, proved that a total destruction of the cord, or a complete transverse lesion had not occurred. I cannot offer any explanation for the facts, but think they should be put on record.

Case II.—Fracture of the seventh cervical vertebra, crushing the first dorsal segment of the cord.

Bruns has recently[2] published a very careful record of a case of traumatic lesion of the spinal cord by a dislocation of the last cervical upon the first dorsal vertebra, destroying

---

[1] It must be remarked that many of Head's statements regarding the reference of visceral pain to surfaces of the body are to be found in this remarkable article of Dana which he has apparently overlooked, and the figure of which is here reproduced. (Fig. 4.)

[2] *Arch. für Psych.*, xxv., 759, 1893.

the first dorsal and lower part of the eighth cervical segments.

The symptoms observed were as follows: complete flaccid paralysis of the legs, with loss of knee and ankle reflexes and of all skin reflexes; paralysis of the body muscles, paralysis of the small muscles of the hands and of the long flexors of the fingers on both sides, with atrophy and reaction of degeneration. Complete anæsthesia up to the second rib and to the spines of the scapulæ; above this, on the body and on the upper extremities hyperæsthesia and pains, but no anæsthesia. (Fig. 5.) Complete paralysis of bladder and rectum, with retention. Purulent cystitis. Priapism. Contraction of pupils and narrowing of the palpebral fissures; bedsores; œdema of the legs, with trophic disturbances of the skin and joints. This condition changed slightly up to the time of the patient's death, which occurred 104 days after the injury; there being a partial return of sensation as low as the fifth rib on the body, a subsidence of the hyperæsthesia of the arms, a return of pupil and lid condition to normal, and an improvement in the motion of the fingers.

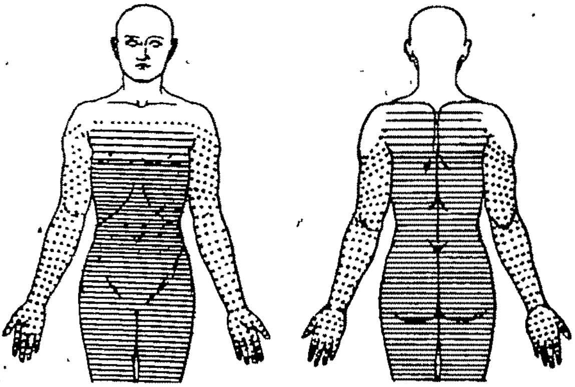


FIG. 5.

Case II.—Bruns. Anæsthesia in lesion of first dorsal.

Remarks.—In view of the fact already established by Reid that the second dorsal segment may lie under the seventh cervical spine; and in view of the fact that recovery of sensation and motion in the arms and hands is inconsistent with a lesion of the eighth cervical segment, I am inclined to suppose that in examining this cord, Bruns has erroneously located the lesion, and that it lay one segment lower than he supposed. This is the only explanation which enables one to bring the facts into harmony with the very numerous cases observed by others.

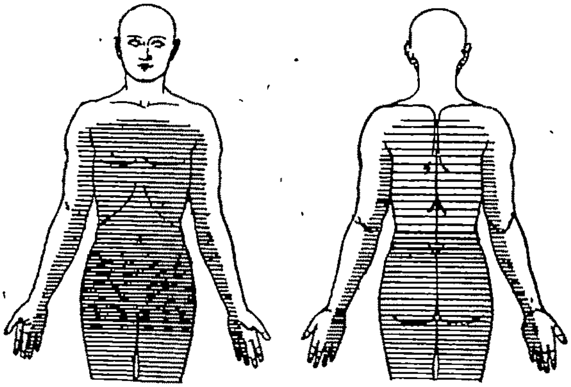


FIG. 6.

Case III.—Bruns. Anæsthesia in lesion of seventh cervical.

Case III.—Hæmorrhagic myelitis after injury to the lower cervical segments. Sensory and motor symptoms.

Bruns reports[1] another case of injury of the back followed by softening, with hæmorrhage in the lower half of the cervical enlargement. The softening involved the seventh and eighth cervical segments in their entire extent; the sixth segment was a little softened in its central region; the first dorsal segment was normal. The symptoms were total paraplegia with loss of knee-jerks, and paralysis of bladder and rectum. The paralysis involved the hands, there being total paralysis of the fingers, and of the long flexors of the wrist and weakness of the long extensors. Pronation and supination were good and elbow motions unimpaired. The anæsthesia was total up to the level of the umbilicus and partial from there to the level of the second rib. (Fig. 6.) All sensations were lost in the ulnar half of the upper extremities on both dorsal and palmar surfaces, while perfectly preserved on the radial half. The patient died a few days after the injury.

---

[1] *Deut. Med. Woch.*, 1889, vol. xv., p. 984.

Case IV.—Fracture dislocation of seventh cervical vertebra, crushing the spinal cord at the eighth cervical segment. Motor and sensory symptoms.

Herter reports[1] a case quite similar to the last one which may be summarised as follows :—A fracture of the seventh cervical vertebra with dislocation forward of the sixth The cord was crushed at the eighth cervical segment and softened at the seventh segment, contained numerous hæmorrhagic foci which extended into the sixth segment being surrounded by inflammatory cells. The fifth segment was fairly healthy, though showing some inflammation. The paralysis in this case involved the legs and body, and the muscles of the

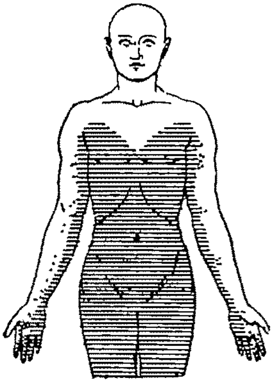


FIG. 7.
Case IV.—Herter. Anæsthesia in lesion of seventh cervical.

---

[1] *Jour. Nerv. and Ment. Dis.*, June, 1891.

hands and forearms, but those of the arms and the supinators and pronators were exempt at the outset. After a few days the pronators were paralysed, and the triceps and biceps became weak. In the paralysed muscles electric reactions were greatly diminished to both currents in five days. Reflexes absent except the cremasteric. The area of anæsthesia, is shown in the diagram. (Fig. 7.) It involved the inner side of both arms and hands, and along its outer limit there was a narrow line of hyperæsthesia.

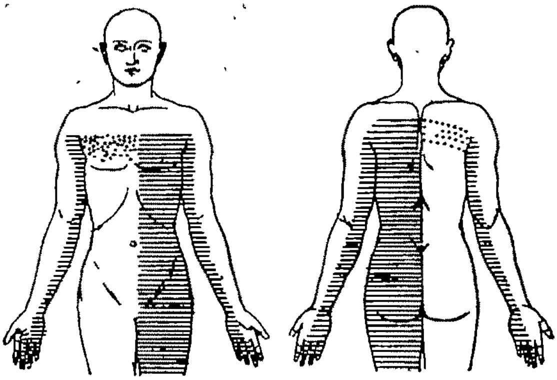


FIG. 8.

Case V.—Williamson. Anæsthesia in lesion of seventh cervical.

Case V.—Myelitis of the cervical region with unilateral sensory disturbances.[1]

A girl 13 years old was suddenly seized with paralysis of arms and legs and of the sphincters during the course of typhoid fever, from which she died within a month of the onset of the paralysis. The symptoms observed were paralysis of all four extremities with the exception of the muscles of the upper part of the arm, deltoid, biceps and spinators being exempt. The knee-jerk was lost upon left side. There was no anæsthesia to touch, but sensations of pain and temperature were entirely lost over the left side of the body up to the level of the third rib, and upon the inner sides of the arms as shown in diagram. (Fig. 8.) There was a small

[1] Williamson, *Lancet*, January 21, 1893.

region of hyperalgesia across the right side of the body and back.

The diagnosis was made of a hæmorrhage in the cord, and from the resemblance of the anæsthetic condition to that occurring in syringomyelitis, it was supposed that the gray matter only was affected.

Autopsy showed an acute myelitis without hæmorrhage in the first dorsal and eighth cervical segments of the cord, especially upon the right side.

Case VI.—Fracture dislocation of the sixth vertebra, crushing the eighth cervical segment. Motor and sensory symptoms.

The following interesting case has been carefully reported by Dr. C. A. Herter.[1]

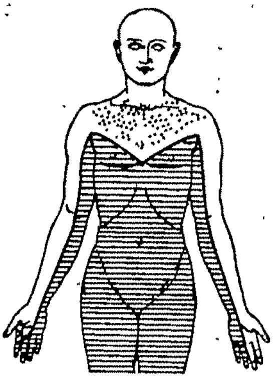


FIG. 9.

Case VI.—Herter. Anæsthesia in lesion of seventh cervical segment.

The patient fell and dislocated the sixth cervical vertebra, which crushed the eighth cervical segment and caused a softening of the seventh cervical segment with hæmorrhage, which extended upward within the central canal to the upper limit of the sixth segment. This condition was found at the autopsy made two days after the accident.

The symptoms immediately after the accident were complete paralysis and anæsthesia, with loss of all reflexes, excepting the left plantar reflex below the level of the armpits, the hands being noticeably weak but not paralysed. Ten hours after admission the condition of sensation was carefully determined by Dr. Herter, and is shown in fig. 9. It will be noticed that on the right side the anæsthesia involved the palmar surface of the little, ring, and middle fingers, and of the third phalanx of the index finger; on the dorsal surface it involved the little and ring fingers with the second and third phalanges of the middle finger. On the left hand the little and ring fingers were involved on both surfaces, and the middle finger on its dorsal surface excluding the first phalanx and the index finger at its dorsal top. The anæsthesia of the body was V-shaped in front and passed across the back at the seventh cervical vertebra. There was hyperæsthesia above this level—in front and behind. Deep and superficial reflexes were absent. There was retention of urine.

---

[1] *Jour. Nerv. and Ment. Dis.;* January, 1890.

Within a few hours of the accident the paralysis had extended and eventually involved the muscles of both hands, the flexors and extensors of both wrists and the triceps. There was slight loss of power in the pronators and supinators, and in the biceps and pectoralis major. Both arms were abducted with forearms flexed and supinated nearly in the position described by Thorburn. The rapid extension of the paralysis and death from respiratory failure were ascribed to the extension of the hæmorrhage upward to the sixth segment. The sensory disturbance, which was the same from the first, was ascribed to the lesion at the seventh cervical segment.

It is not necessary to cite further cases,[1] although a number collected by Thorburn,[2] and by Chipault[3] and by White[4] might be offered as additional proof. It is to be

---

[1] The following cases may be referred to:—Bastian, *Med. Chir. Trans.*, London, 1890, vol. lxxiii., p. 152; Raymond, *Revue Neurologique*, 1893, Nos. 5 and 6; George Preston, *Med. News*, 1893, March 18; Hans Daxenberger, *Deut. Zeitschr. für Nervenheilk*, 1893, iv.; Kraus, *Zeitschr, für Klin. Med.*, xviii., 343.

[2] "Surgery of the Spinal Cord."

[3] "La Chirurgie Médullaire."

[4] "The Surgery of the Spine."

noticed that in none of these cases did the lesion destroy the sixth dorsal segment. In none of these cases was the outer surface of the hand and thumb and arm invaded by the anæsthesia. In the following cases the sixth dorsal segment was destroyed.

Case VII.—Dislocation and fracture of sixth cervical vertebra, crushing the cervical cord. Hæmorrhage in the cord. Sensory and motor symptoms.

F. H., aged 46, was brought to the Presbyterian Hospital on November 19, 1892, suffering from a fracture of the sixth cervical vertebra, due to a fall. There was tenderness and depression over the sixth and seventh spines and a projection in the pharynx. He was totally paralysed below the waist, and his arms were partially paralyzed. His urine was retained. On the next day, when I examined him, the following condition was present.

Motion.—Patient could not move from the dorsal decubitus and attempts to move his head were too painful and dangerous to be repeated. His arms were abducted, forearms flexed and hands pronated, lying on his chest, in the position described by Thorburn as characteristic of lower cervical lesions. Voluntary acts to produce these positions could be performed. He could not extend his forearms, but could flex and supinate them; all motions in the wrists and fingers were impossible upon the left side and on the right side were very feeble, there being a slight flexion and extension of the long muscles, but none of the intrinsic muscles of the hands.

There was total paralysis of the respiratory thoracic muscles, all respiration being diaphragmatic. He could not move his abdominal muscles. His legs were totally paralyzed and relaxed.

Reflexes.—Epigastric reflex absent. Abdominal and cremasteric reflexes present and normal. Knee-jerks slightly exaggerated. No ankle clonus.

Sensation.—There was total loss of all sensations in front and at the sides up to a line about the body at the level of the seventh rib, and very marked diminution of sensations up to the level of the nipples, gradually merging into normal sensation at the level of the third intercostal space. It was not possible to test his sensation on the back, as he could not be moved. (Fig. 10.) The diminution of sensibility to all stimuli was quite well marked upon the arms, its extent being less on the right arm than on the left one. The inner side of both arms and the entire hands were quite anæsthetic, but the outer side of the forearms and arms seemed to have preserved some sensibility above the wrists, this being much more acute on the right arm and forearm than on the left one. It was not until the deltoid region was reached on the left arm that he felt cotton wool as keenly as upon his face. There did not appear to be any well defined region of hyperæsthesia; yet over the region on the chest above the line of diminished sensibility he seemed to feel pain more acutely than on the face. Touch, pain, and temperature senses were equally impaired.

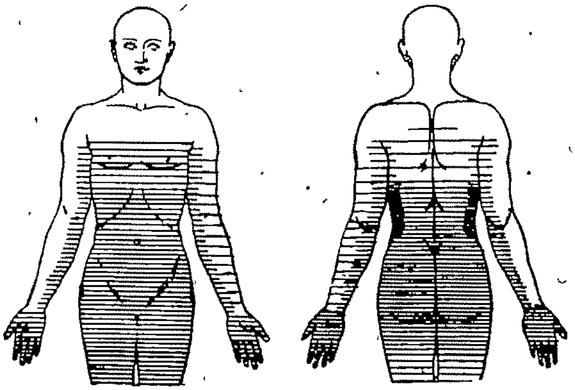
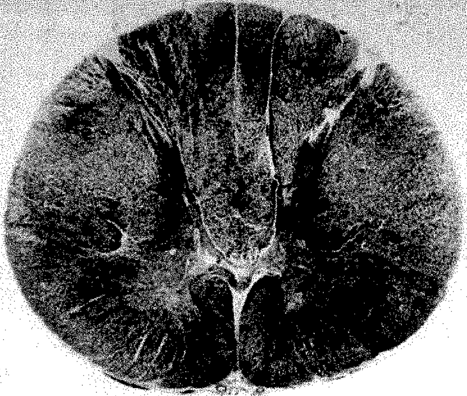


FIG. 10.

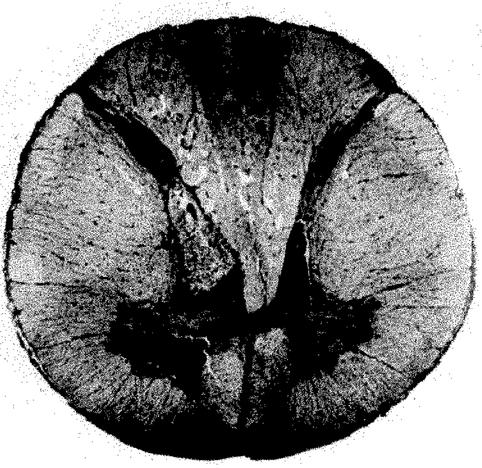
Case VII.—Starr. Anæsthesia in lesion of sixth cervical.

Bladder and rectum were not under voluntary control. The urine was retained and had to be drawn by catheter. Partial priapism existed. No attempt was made to empty his rectum.

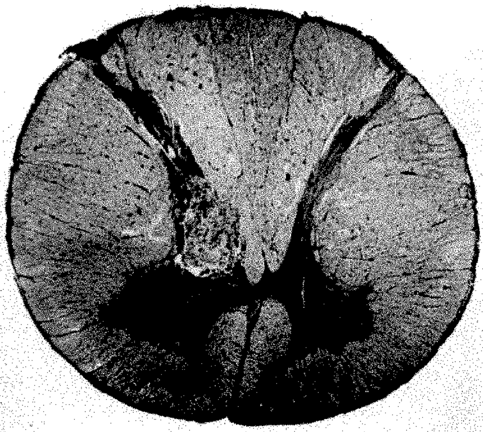
PLATE I.



Cerv. V.

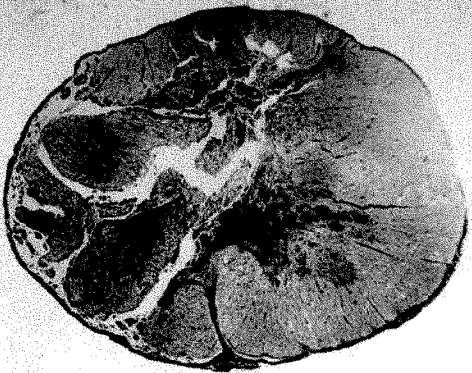


Cerv. VI.

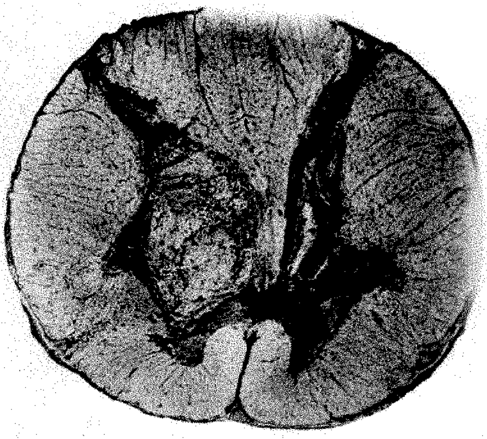


Cerv. VII.

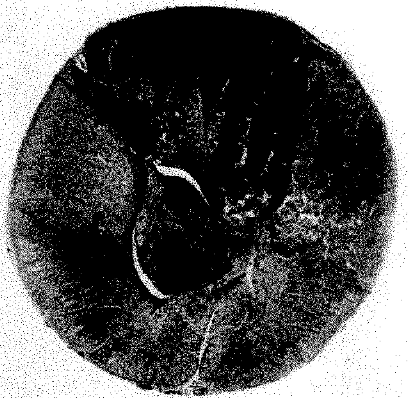
PLATE II.



Cerv. VIII.



Dors. I.



Dors. II.

Collotype by Danielsson & Co., London.

The pupils reacted slowly to light and in accommodation, but did not dilate when the neck was irritated. The lids were not as widely opened as they should have been. It was thought that the cilio-spinal centre had been affected.

His mind was clear up to the time of his death, which occurred about thirty-six hours after the injury; his condition remaining the same. An attempt was made to operate, but the change of position produced such disturbance of respiration that it was abandoned. The following account of the autopsy was kindly furnished me by the pathologist, Dr. John K. Thacher, and the appearance of the cord is shown in the plates made from photographs of sections prepared by Dr. Power. (Plates I. and II.)

On dissection of the vertebral column, the laminæ of the sixth cervical vertebra were found fractured, and there was a dislocation forward of the vertebral column between the fifth and sixth spinous processes, and between the bodies of the sixth and seventh vertebræ. At this point the cord was compressed and softened. Microscopical examination showed much crushing and hæmorrhage. The whole of the left half of the eighth segment was broken up, as well as the right posterior column except its posterior edge. The fibres and cells were destroyed, and there were many hæmorrhages, especially in the gray matter. The gray matter of the right side was broken up, especially at the anterior part of the posterior horn and showed some extravasation of blood. But the tip of the posterior horn and some of the anterior portions of the anterior horn appeared uninjured, as did also the anterior and lateral columns of white matter.

Passing upwards from the segment crushed, to the seventh segment, a spot of disintegration and hæmorrhage was found involving the anterior half of the left posterior horn and a little of the adjacent anterior horn and lateral and posterior white column. The hæmorrhage also extended in a narrow line along the outer edge of the posterior horn nearly to the surface of the cord.

In the sixth segment the same appearances were found, but in the fifth the tissues were normal.

Passing downwards to the dorsal region of the cord, in the first dorsal segment a displaced mass of white matter was noticed replacing most of the gray matter of the left side and surrounded by a zone of hæmorrhage. On the right side there were numerous hæmorrhages scattered through the gray matter, and apparently a small displaced mass of white matter in the base of the posterior horn.

In the second dorsal segment the lesion on the left side was similar to that in the previous segments, but a little smaller. On the right side no hæmorrhages were seen in the anterior horn, but in the posterior horn was a long narrow oval hæmorrhage running nearly the entire length of the horn.

In the third dorsal segment no lesion was to be seen on the left side, but in the right posterior horn the same hæmorrhage found in the preceding segment was again found, though smaller.

Below the third dorsal segment the cord appeared to be normal.

Summary.—The lesion, therefore, consisted of a disintegration of the cord at the eighth cervical segment and a hæmorrhage in the two segments above and below this, especially on the left side of the cord.

In regard to the area of anæsthesia upon the body it is to be noticed that it was much below the level of the lesion. Sensations were felt in slight degree above the level of the seventh rib; they were fairly well felt above the nipples, they were quite normally felt above the third rib. This preservation of sensation upon the body at a level some distance below the lesion, is not unusual. I have careful records of several cases of transverse myelitis in the dorsal region with autopsies in which this condition has been noticed. And the cases reported by Church and Eisendrath[1] may be cited in confirmation of the fact. It is noticed that, except on the ulnar side of the forearms and arms and in the hands, the anæsthesia in my case was not total. The hands were both totally anæsthetic.

---

[1] *Am. Journ. Med. Sci.*, 1892.

Case VIII. — Caries of cervical vertebra. Brown-Séquard paralysis. Death.

A girl of 15, previously in good health, noticed in August that her neck was becoming stiff and painful on motion. The pain was in the course of the great occipital nerves, and by October her head had become so rigid that no active or passive motion of the neck was possible. During October weakness in the arms, especially in the left arm, began, and soon the left leg also became weak. She was sent to my clinic on October 22, and then presented the following symptoms. The head was held rigid, turned slightly to the right, the chin slightly elevated, and passive motion was prevented by the rigidity of the deep muscles of the neck as well as by a deformity of the spine, which was evident both by a deep hollow in the neck at the fourth and fifth cervical vertebræ and by a projection at the back of the pharynx. Pain in the neck and over the back of the head was a prominent symptom, but was not associated with anæsthesia. The left upper extremity was partially paralysed, all movements being very much impaired; those at the shoulder and elbow joints more than those at the wrist and fingers. No motion of the arm was possible, it hanging helpless. Motions of the forearm were very weak, extension being impossible; the forearm was semiflexed upon the arm. While all the fingers could be moved, she had no grip. The triceps and wrist tendon reflexes were greatly exaggerated, a triceps clonus being obtained. All sensations were perceived equally in the left side and in the face; and the muscular sense was keen and quick.

The left lower extremity was weak, both at hip, knee, and ankle joints, and the tendon reflexes were much exaggerated. The sensation was normal; that is, the same as in the face.

The right upper extremity was freely moved, but was markedly anæsthetic up to the shoulder. Tactile sensations, sensations of pain, heat, and cold were not perceived by any means as promptly, as accurately, or as intensely as in the corresponding part of the opposite limb. There was marked ataxia of movement in the right hand when her eyes were closed: and the perception of its position was less rapid and accurate than on the left side. There was no motor paralysis and no increase of tendon reflexes.

The right lower extremity was not paralysed, the knee-jerk was normal, no ankle clonus was obtained. The same kinds of anæsthesia were found as on the right arms, though sensations were rather better in the lower than in the upper extremity. The area of anæsthesia is shown in fig. 11.

The gait was much impaired. She staggered in standing and walking, with a tendency to fall toward the right when her eyes were closed, and her gait was decidedly ataxic.

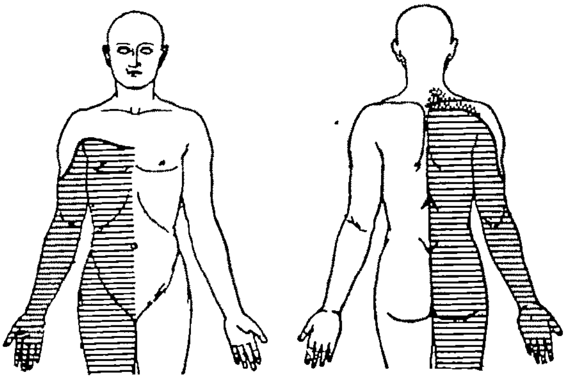


FIG. 11.

Case VIII.—Starr. Anæsthesia in lesion of fifth cervical segment.

The control of the sphincters was somewhat impaired. She had to wait a long time before her water would pass, and a loss of voluntary control of the bladder soon ensued, so that a catheter had to be used.

There was no hyperæsthesia of the paralysed side and no band of anæsthesia about the trunk on that side.

By the end of November, all of the symptoms had increased in intensity, the paralysis of motion of the left side being complete, and the anæsthesia on the right side being increased; she was unable to sit up or to control her sphincters. She had distressing attacks of dyspnœa.

The condition finally became one of total paraplegia, and in January she died.

Diagnosis.—It was evident from the first that a caries of the fourth and fifth cervical vertebræ had resulted in a displacement of these bones, which had produced pressure upon the left half of the spinal cord at the level of the fifth and sixth cervical segments. That these segments or their nerves were seriously implicated was evident from the fact that a marked atrophy, as well as paralysis and a reaction of degeneration, was found to exist in the deltoid, biceps, triceps, supinator longus, supra and infra spinatus, and pectoral muscles of the left arm, while no such atrophy or reaction was found in the other muscles in spite of their paralysis. In the right arm the anæsthesia was more marked over the back of the shoulder and arm and on the outer side of the arm and forearm than elsewhere. The pressure was sufficient to cut off, to some extent, motor impulses downward to the left leg on the side diseased, and sensory impressions of all kinds from the right side of the body below the level of the lesion.

Operative interference appeared to be impossible, and the disease made rapid progress, producing finally total paraplegia and death from respiratory paralysis. An autopsy was not made.

With the cases already cited it would easily be possible to lay down certain districts of skin upon the upper extremity, and assign each district to its segment of the cervical cord. It would be possible to cite also numerous cases of stab wounds of the cord and of tumours of the cord which confirm these conclusions.[1] But did we attempt this the objection might reasonably be raised that from traumatic cases where shock cannot be excluded and where temporary effects cannot be wholly distinguished from permanent effects such conclusions would be unwarranted.

The following case, however, is not open to this objection, and appears to afford exactly such information as is needed to enable an exact localisation to be reached.

---

[1] Incised wounds of the spinal cord. Neumann, *Virchow's Arch.*, cxxii. h. 3. Sachs, *Jour. Nerv. and Ment. Dis.*, xiii., November, 1886.

It is a case of sarcoma growing upon and invading the cord upon one side, beginning at the lower cervical and first dorsal segments, and extending gradually to other segments. As the sarcoma grew the anæsthesia increased in extent.

Case IX.—Tumour of the lower cervical region of the cord with progressive anæsthesia.[1]

Male, 29, began to suffer in May, 1891, from numbness in the ulnar side of the right little finger. The numbness, which was attended by actual anæsthesia, gradually extended to the entire little finger, then to the ring finger and then to the ulnar side of the middle finger. As it extended to the ring and middle fingers it extended to the ulnar surfaces of the hand, and then gradually up the ulnar surfaces of the forearm and arm, to the axilla which were anæsthetic in August, 1891. Later, the anæsthesia increased upon the hand, gradually reaching the thumb, and then including it; and finally advanced up the radial border of the forearm and arm to the insertion of the deltoid, at which point it ceased about thirteen months after the onset. Parallel with this extension of sensory loss there was a gradual extension of paralysis with atrophy and reaction of degeneration. The paralysis began in the interossei and hypothenar eminence, then invaded the thenar eminence, then the long flexors of the fingers, and then all the muscles of the forearm except the supinator. The muscles became rigid before they were paralysed, but relaxed again as degeneration set in. In the course of the year all the muscles of the arm became involved and paralysed except the supinator longus and the deltoid; the pectoralis major and shoulder muscles became rigid. From the first it was noticed that the right palpebral fissure was small, that the right lid drooped and that the right pupil was small.

In September, 1892, the patient died, a large sarcoma having developed in the chest.

---

[1] Ross and Herter, *New York Medical Record*, August 12, 1893. It is interesting to note that though the tumour was confined to one side of the cord the symptoms were not those of Brown-Sequard paralysis. The same fact was noted in a case of spinal cord tumour seen by me with Sachs, and fully reported by him.

The autopsy showed a small sarcoma lying upon and invading the spinal cord on the right side, extending up as high as the fifth cervical segment and as low as the third dorsal segment, but being largest at the lowest cervical and first dorsal level.

A somewhat similar case has been recently reported by Murray and Johnson,[1] and several others might be cited of either tumour or myelitis advancing up the cervical cord and producing a gradual extension, both of paralysis and of anæsthesia. As the symptoms, however, in all these cases are identical, it does not seem worth while to cite them in full.

There are certain other facts which may be mentioned as affording confirmatory evidence of the areas of skin corresponding to the segments of the cervical enlargement. It is well-known to everyone who has watched the progress of cases of locomotor ataxia of the ordinary progressive type that as the disease ascends the cord the numbness and pain begin in the upper extremity in the little finger, then extend to the ring finger and then up the inner border of the hand and forearm. Finally the entire hand becomes numb and the thumb is the last to be ataxic. Thus the extension of the sensory symptoms in this disease corresponds quite exactly with that which has just been described in the case of cervical tumour.

There is also a disease which quite commonly invades the upper part of the cervical cord, viz.: chronic hypertrophic pachymeningitis. It is to be remembered that in this disease the pain is felt chiefly in the neck over the tops of the shoulders and in the outer side of the arms, and only rarely in the forearms and hands. The characteristic position of the hand and wrist pictured by Charcot[2] is explained by him as due to contracture of the muscles irritated by invasion of the nerves from above downward, the radial and median being affected, the ulnar escaping. Thus we see the exact converse of the picture presented by the advance of the disease locomotor ataxia.

---

[1] *Medico-Chirurgical Transactions*, vol. lxxv., p. 47, 1892.

[2] *Leçons sur les Mal. du Syst. Nerv.*, vol. ii., p. 251.

Conclusions.

There are a number of interesting facts to be drawn from the study of the cases here collected.

First, as to the distribution of anæsthesia in lesions of the cervical enlargement of the spinal cord. By combining the diagrams already given with numerous other diagrams published by Thorburn and others, and comparing the results with descriptions of numerous cases collected by Chipault, White and other surgeons, it is possible to produce a schematic diagram such as is here presented demon-

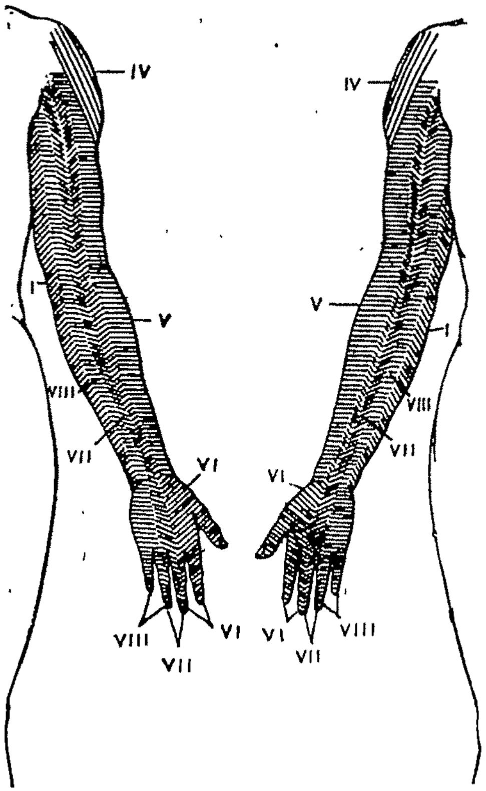


Fig. 12.

Diagram to show the area of skin upon the upper extremity related to the different segments of the spinal cord from first dorsal to fourth cervical.

strating the areas of skin upon the arm which are related to the various segments of the cord between the second dorsal and the fourth cervical. (Fig. 12.)

(1) The first area is a narrow one upon the inner side of the arm and forearm extending down to the wrist but not involving the hand. This includes a small zone both upon the dorsal and palmar surfaces of the arm and forearm, but is quite narrow. While it is probable that the upper portion of this area in the axilla is related to the second dorsal nerve, as Head maintains, I can find no exact lesion of the cord limited to the second dorsal segment and associated with such a limited anæsthesia to prove this statement.

There is a case reported by Charcot[1] in which a bullet wound involved the first dorsal nerve only, and in which a small area in the axilla remained sensitive; the other parts included in area number one being anæsthetic, but as no examination of the cord was made in this case the conclusion drawn from it is of no more weight than that which might be drawn from a knowledge of the anatomy of the intercostal humeral nerve. Area number one corresponds to the first dorsal segment.

(2) The second area includes the ulnar side of the hand, both palmar and dorsal surfaces, the little finger and one half of the ring finger. It also includes a narrow strip of the skin upon the dorsal and palmar surfaces of the arm up to the axilla. This is proved by the fact that when lesions invade the eighth cervical segment of the cord the zone of anæsthesia upon the front and back of the arm and forearm is decidedly wider than when the first area alone is anæsthetic. It affords also a confirmation of the statements of Sherrington that there are no isolated patches of skin entirely separated from the body related to the sensory nerves. The second zone corresponds to the eighth cervical segment.

(3) The third zone extends to the middle line of the middle finger, includes the middle of the palm and back of the hand, and runs up the centre of the forearm and arm on both surfaces. This corresponds to the seventh cervical segment.

---

[1] *Arch. de Neurol.*, 1891.

(4) The fourth zone includes the remaining skin of the fingers and thumb and of the hand as high as the wrist and a narrow strip of skin up the forearm and arm on both surfaces to the axilla. This corresponds to the sixth cervical segment.

(5) The fifth zone includes the skin upon the outer side of the forearm and arm on both surfaces as high as the insertion of the deltoid muscle. This corresponds to the fifth cervical segment.

(6) The fact that in lesions as high as the fifth segment hyperæsthesia occasionally occurs over the top of the shoulder and in the region of the supra-clavicular nerves, leads us to believe that the fourth cervical segment is in relation with this area. But inasmuch as lesions of the fourth segment of a destructive nature are uniformly fatal no case of anæsthesia can be found to determine the exact extent of skin which corresponds respectively to the fourth and third cervical segments.

It is hardly necessary to point out the differences between the areas of anæsthesia produced by cord lesions and those produced by lesions of individual nerve trunks, although a glance at the diagram and its comparison with Flower's well-known plates show this immediately. It is also evident that a contrast of the anæsthesia occurring in spinal cord lesions with that occurring in multiple neuritis affords an important point of diagnosis between these two conditions. It will be remembered that in multiple neuritis, the anæsthesia affects all the fingers simultaneously, then the entire hand, and then extends up the arm, involving all surfaces on both sides in an area which has been aptly described as the "glove area" corresponding as it does to the region of skin covered by a glove of various lengths. I have never seen a case of multiple neuritis in which the anæsthesia extended as high as into the axilla. It is usually limited by a line drawn around the elbow or a line in the middle of the arm.

Lastly, the area of anæsthesia occurring in hysterical conditions is different from that described as occurring in spinal lesions, for there is never the patch of normal sensitive skin over the top of the shoulder and down the outer side of the arm to the insertion of the deltoid such as we have seen in all the cases of spinal lesions; and in hysterical anæsthesia it is well known that the neck is usually anæsthetic.

Clinical observers in the future should attempt by the aid of diagrams to define a little more exactly the region of the anæsthesia in the arm and should also seek for a narrow zone of hyperæsthesia separating the normal skin from the insensitive region, which zone will necessarily be very narrow in the upper extremity. In discussing the anæsthesia from lumbar lesions, I was able to show that such a zone of hyperæsthesia often existed of a most irregular shape in unexpected areas which were easily explained when the exact region of the skin, corresponding to the segment just above the lesion was determined.[1]

Secondly, lesions in the lower cervical region of the cord produce a number of symptoms due to a lesion of the ciliospinal centre which have been frequently overlooked by observers. The situation of this centre is in the first dorsal segment of the cord, and according to Klumpe,[2] the symptoms are only produced by a lesion of the cord, or of its nerve roots, not by a lesion of the nerves after they have issued from the spinal canal and formed the brachial plexus.

The symptoms alluded to are myosis, with sluggish reaction of the pupil both to light and in accommodation, a decided narrowing of the palpebral fissure, so that the eye-ball looks smaller than normal, and a slight retraction of the eye-ball. There is no flattening of the face, nor are there any vaso-motor symptoms such as may be produced by a lesion in the course of the cervical sympathetic nerve. These symptoms are very easily overlooked if they are present upon both sides. But when the lesion has been unilateral they have been noted by a number of observers.

Kraus[3] has described them in a number of cases of traumatic injury of the cervical cord due to fracture of the seventh cervical vertebra.

---

[1] *L. c.*, see Cases V. IX. and XII.

[2] Klumpe, *Rev. de Med.*, 1885, No. 7 and 9.

[3] Kraus, *Zeitsch. für Klin. Med.*, vol. xviii.

In a case of unilateral compression of the cord by a fracture of the seventh cervical vertebra which I recently saw with Dr. Bryant, at St. Vincent's Hospital, these symptoms were very noticeable upon the left side. I believe with Kraus that a more careful observation will reveal the existence of these symptoms on both sides in all cases of a crush of the lower part of the cervical and upper dorsal region.

Thirdly, a careful study of the cases here collected furnishes additional evidence in regard to the localisation of the motor functions of the spinal cord. This serves to confirm very fully statements made by me in 1884,[1] and again in 1888,[2] in regard to the representation of the various muscles of the upper extremities in different segments of the cervical region.

In the articles alluded to, it will be found that every muscle is assigned a representation in more than one segment of the cord, and this fact has been accepted by Mills[3] and by Kaiser.[4] It is well-known that the groups of cells in the anterior horns of the cord have a considerable extent vertically, so that some groups are long enough to reach through two or even three segments and but very few groups can be said to be situated in one segment only. But inasmuch as these groups of cells govern individual muscles, as we must believe in view of the facts known regarding anterior poliomyelitis, it is evidently a mistake to assign the representation of single muscles to single segments. I cannot therefore adopt the arrangement proposed by Thorburn[5] and copied by Collins,[6] in which individual muscles are assigned to single segments.

If cases such as these here collected are carefully studied it will be found that a lesion limited to one segment rarely paralyses any muscle completely. If, however, the lesion

---

[1] *Amer. Jour. of Neurology*, August, 1884.

[2] *Amer. Jour. Med. Sci.*, May, 1888.

[3] C. K. Mills, *Therapeutic Gazette*, May and June, 1889.

[4] O. Kaiser, *Die Functionen der Ganglienzellen des Halsmarks*, Haag, 1891.

[5] *Loc. cit.*

[6] *New York Medical Journal*, February, 1894.

extends through two or more segments the paralysis is complete. Or, if a lesion is a progressive one extending either upward or downward, it will be found that the muscles will be paralysed in greater and greater degree as the lesion extends. These facts are only to be explained upon the theory that individual muscles have a considerable vertical representation in the cord, and this as we have already seen corresponds to the anatomy of the groups of cells. The following table, therefore, presents the facts in an accurate manner.

I am quite prepared to admit that the vertical extent of individual groups of cells may vary in different individuals. Thus in cases carefully studied by Beevor and by Johnson, the lesion, though involving the sixth cervical segment, caused no paralysis of the biceps; while on the other hand, in one of Herter's cases the biceps was decidedly involved by a lesion of the sixth segment. It is, therefore, probable that the representation of muscles is not always the same in different cords. The table, however, may be considered as fairly accurate because it combines a large number of facts derived from the study of very many cases. It is, perhaps, more accurate than the earlier tables. In recent cases care has been taken to examine the paralysed muscles electrically, and the existence of the reaction of the degeneration is certainly a positive proof that the groups of cells governing the muscles have been destroyed.

In conclusion, I think it may be stated that the sensory and motor functions of the cervical region are now as well determined as those of the lumbo-sacral region of the spinal cord.

The plates show the disintegration of the spinal cord at the eighth cervical segment, and the hæmorrhage into the cord in the segments above and below it in Case VII.

Table Showing the Muscles Represented in Groups of Cells in the Various Cervical Segments of the Spinal Cord.

IVc.	Vc.	VIc.	VIIc.	VIIIc.	Id.
Diaphragm. Lev. Ang. Scap. Rhomboid. Supra & Infra Spin. Deltoid. Supin. long. Biceps.	Rhomboid. Supra & Infra Spin. Deltoid. Supin. long. Biceps. Supin. brev. Serratus mag. Pect. (clav.) Teres minor.	Biceps. Serratus mag. Pect. (clav.) Pronators. Triceps. Brach. Ant. Long Extensors of Wrist & Fingers.	Pronators. Triceps. Brach. Ant. Long Extensors of Wrist & Fingers. Pect. (costal). Latis. dorsi. Teres major. Long Flexors of Wrist & Fingers.	Long Flexors of Wrist & Fingers. Extensor of Thumb. Intrinsic Muscles of Hands.	Extensor of Thumb. Intrinsic Muscles of Hands.

Reviews.

(I.)—*Klinische und anatomische Studien über die Pellagra.* Von Dr. Franz Tuczek, Berlin, Kornfeld, 1893.

(II.)—*Arbeiten aus dem Institut für Anatomie und Physiologie des Centralnervensystems, an der Wiener Universitat.* 2 vols. Edited by Prof. Obersteiner, Leipzig, Deuticke, 1892-1894.

(I.)—This book, of over a hundred pages of letterpress, and ten pages of plates, is, as the author informs us in the preface, the result of a tour of investigation through the pellagrous districts of Italy. Its avowed object is partly to excite the interest of the writer's German colleagues, especially neuropathologists, partly to contribute something to the morbid anatomy of pellagra. But we venture to think that the book will appeal to a wider audience than the author in his modesty aims at, for it is marked by intimate personal knowledge of the subject, having a succinctness to which completeness is not sacrificed, and it is enriched with some suggestive remarks on the interesting class of toxic diseases of which pellagra is now, on apparently quite sufficient grounds, believed to be an example. It will be enough here briefly to indicate the nature and arrangement of the contents.

After a few pages of introduction in which toxic diseases and their selective action on different parts of the nervous system are dealt with, a short historical account is given of the first appearance of pellagra, and its gradual diffusion from the province in Spain in which it arose over Italy the South-west of France, Roumania, and Corfu, its ravages so far being almost confined to European countries, and becoming, in some parts of Italy, nothing less than a national calamity. This will be appreciated from the fact that in 1884, over ten thousand cases of pellagra were treated in Italian hospitals and asylums. The etiology of the condition seems to be clearly traceable to the use of maize, but this will only produce it if it is damaged in some way. In the Italian domicile of the disease, the maize often does not come to maturity and is therefore easily exposed to damage; its use in this damaged state apparently gives rise to the disease. This is made more evident by the fact that it prevails mostly in years of bad harvest, in which the corn is cut before it is ripe, and is gathered while still damp. It is supposed that in this damaged state some substance is produced which acts as a poison to the nervous system, and it is significant that a spirit distilled from this damaged maize can also evoke the symptoms of pellagra. The various predisposing causes are next briefly alluded to, and the different forms in which the disease manifests itself. Its various stages are also considered, the first, in which the symptoms are mainly premonitory, a second characterized mainly by cerebral or spinal disturbances and psychical changes, and a third or cachetic state with its various phases. The rest of the book is devoted to the setting out of the clinical symptoms of numerous cases and the morbid anatomy of several. In conclusion, the curiously symmetrical changes in the spinal cord are described, affecting both lateral and posterior tracts, and closely resembling the condition of the cord recently described in association with anæmia. These changes are figured in a series of well executed plates, while a successful collotype representation of the condition of the skin which gives the name to the disease, and a map showing its distribution in Italy, give completeness to a most excellent monograph.

JAMES TAYLOR, M.D.

(II.)—These volumes contain a number of valuable contributions to the anatomy and pathology of the nervous system. Dr. Emil Redlich has an exhaustive article "On the Posterior Roots of the Spinal Cord and the Pathological Anatomy of Tabes Dorsalis," and his conclusions, that the first parts to be affected are the posterior roots within the spinal cord confirm the observations of Leyden, Marie, Takács and Dejerine. The determination of the exact starting point of the degenerative process has been made the subject of special inquiry by Redlich and Obersteiner in a paper "On the Nature and Pathogenesis of the Tabetic Degeneration of the Posterior Columns." By making oblique sections so as to cut the posterior root in the direction of its entrance into the spinal cord they find that the pia mater causes, in the normal state, a considerable constriction of the roots, the constriction being caused for the most part by the circular fibres of the pia, one or two vessels also taking part in its formation. Between the constriction and the cord itself the fibres of the posterior roots lose their medullary sheaths for a short distance and the axis cylinders are unprotected.

In locomotor ataxia, even in early cases, the roots on the spinal side of the constriction are degenerated. The cause of this degeneration is a chronic hyperplasia of the connective tissue of the pia mater which forms the constriction. This presses on the nerve fibres, and leads to degeneration. As the fibres which go to Lissauer's boundary zone lie peripherally in the posterior roots, it becomes apparent why the boundary zone suffers so early in this disease. The degeneration of the posterior roots outside the cord is probably due to the same cause, and the degenerative process may even extend to the ganglion cells of the spinal ganglia, as has been shown by Raymond. The tendency of syphilis to produce a chronic hyperplasia of connective tissues, readily explains the frequency of locomotor ataxia as a sequela of this disease. Instead of the chronic inflammation of the pia, a gummatous infiltration may occur without necessarily leading to degeneration of the nerve fibres (syphilitic pseudotabes).

The pathological anatomy of *paralysis agitans* is also treated by Redlich, and his observations confirm in the main those of Koller and Ketscher. He distinguishes between purely senile changes, such as blocking of the central canal and pigmentation of the ganglion cells, and changes which are characteristic of the disease. These occur in the spinal cord and medulla, and begin as endo- and periarteritis. This leads to sclerosis of the connective tissue of the cord, and indirectly to degeneration of the nerve fibres. The cervical and lumbar enlargements are the parts most affected, especially the posterior columns and crossed pyramidal tracts. The greater affection of the cervical region may probably explain the characteristic rigidity of the upper limb and neck muscles; while periarterial sclerotic foci in the medulla might cause the characteristic disturbance of speech. For further details, however, reference should be made to the original papers.

WILLIAM BULLOCH, M.B.

BRAIN.

PART IV., 1894.

Original Articles and Clinical Cases.

THE FINER ANATOMY OF THE INFUNDIBULAR REGION OF THE CEREBRUM INCLUDING THE PITUITARY GLAND.

BY HENRY J. BERKLEY, M.D., BALTIMORE.

From the Pathological Laboratory of the Johns Hopkins University and Hospital.

Anatomy.—The material used in this study was obtained from the brains of twelve dogs of various races, and six mice of the gray variety. The results obtained from the dog were on the whole more satisfactory than those from the rodent, though, in some instances, the tingeing of the nerve elements was finer with the smaller animal. All examples of both species were *adults,* and some of our best preparations of the neuroglia elements were had from the infundibulum of a very old dog of the setter breed.

The region examined, began anteriorly at the posterior margin of the chiasma opticorum, and included in the first sections some of the fibres of the optic nerves, then passing dorsally, the anterior portion of the infundibulum, the infundibulum proper, the tuber cinerium, the anterior and posterior lobes of the pituitary gland, and the corpora mammillaria as far as their posterior border.

All sections were made vertical to the surface of the brain, it being found that these gave the best results; and in the central region of the infundibulum, where it opens into the third ventricle, the sections took in an area as high as five millimetres above the floor of the inferior surface of the cerebrum. Laterally, the area of the sections was confined to the margins of the gray matter of the temporal convolutions, but did not extend into their substance. In all, about 2,500 sections were examined.

The staining agents used were the rapid silver method for the nerve elements, and my picric acid modification of the same, for the neuroglia elements and nerve cells. The magenta method of Nissl, eosin-hæmatoxylin, and a number of the aniline dyes were used for confirmatory purposes only.

I.—THE PITUITARY GLAND.

Our work commenced by a study of the pituitary body, as it is best seen in vertical section after it has been removed from the basis cerebri with a portion of the tuber cinerium attached. Sections were also made of the inter-thalamic region with the hypophysis connected, in order to ascertain the relations of the nerve elements of the infundibulum and those of the nervous portion of the gland, and to preserve as far as possible the landmarks, as the Golgi method, owing to its characteristic action in staining only a portion of the nerve fibres and cells, also owing to the overlapping of the white and gray substances, renders it very difficult to keep track of the intermingling ganglionic regions.

In the mouse, the pituitary body proved to be too small to obtain satisfactory sections, not being larger than the point of a good-sized pin. In the dog it is about the size and shape of a small cherry, and is divided into two unequal portions by a closed ventricle. The posterior portion, the *lobus infundibuli*, which is directly attached to the infundibulum, is a rounded oval in form, and varies both in shape and size but slightly in the various animals used. This lobe is so strongly adherent to the dura that it pulls out of the rest of the pituitary body in removing this with the brain, unless the membrane is dissected with it from the base of the skull.

The second half of the organ, the anterior or glandular portion, is attached loosely to the rounded base of the infundibulum, by connective tissue and a large number of vessels, both veins and arteries, that directly pass into it from the substance of the infundibulum, and to the *lobus infundibuli*, by connective tissue and large vessels. This glandular portion of the body may be roughly divided into two rounded lateral thicker portions, united by a central thinner portion, into which the lobus infundibuli is pressed. Between the anterior and posterior lobes, is a constant closed cavity. The wings of the main cavity have horns, which pass far outward toward the inferior and lower margins of the lateral thickenings of this lobe. These cavities are lined throughout their extent by cylindrical epithelium. Besides the main cavity, a number of smaller cavities are to be found, both in the nervous and glandular portions of the organ, usually lined with cylindrical epithelium, but sometimes, especially along the margin of the body, other cavities of a different nature are to be observed, evidently formed by the coalescence of two or more follicles, and are commonly filled with a colloid material.

For the general histological features of the glandular elements of the anterior lobe, I must make reference to the text book of W. Krause,[1] the monographs of Lothringer,[2] Schonemann,[3] and Stieda,[4] and for histology and development, to the very excellent work of W. Muller,[5] as our researches only concern the intimate nerve distribution.

The Glandular Portion of the Hypophysis.—In the glandular portion of the body, nerves, other than those belonging to the sympathetic system, are not found. They are very fine varicose fibres, with numerous ramifications and ultimate branchlets coming off from the main stems at a right or slightly obtuse angle. As in all the other secretory organs we have studied, bundles of small nerves are to be seen following the course of the arteries, and in the present instance these vascular bundles do not seem to be unusually numerous. From them come off, at irregular intervals, single fibres, or branches from the main stems, which pursue a very irregular course through the glandular substance, crossing over or accompanying the large venous channels in the septa, and finally being distributed upon the coils of the epithelial cells forming the follicles. As in the illustration (fig. 1), single nerve fibres may frequently be seen coming from some considerable distance over the surfaces of the follicles, across veins and septa, and finally breaking up into a terminal figure, with quite numerous ball-shaped endings, which appear to lie in the inter-cellular substance; the whole arrangement being very similar to that of the adrenal gland. No nerve cells are to be found in the substance of the organ, and all nerves belonging to it appear to be derived from branches of the carotid sympathetic plexus.

---

[1] W. Krause, *Handbuch der Anatomie.*

[2] Lothringer, *Arch. fur Mik. Anat.*, Bd. xviii.

[3] Schonemann, *Virchow's Arch.*, Bd. cxxix.

[4] Stieda, *Inaug. Dis. Konigsberg*, 1889.

[5] W. Muller, *Jenasche Zeitschrift*, Bd. vi., 1871.

The Infundibular Lobe of the Pituitary Gland.—The microscopic appearances of the very numerous sections we have made of this portion of the organ are somewhat at variance with the ideas received from the text books, in respect to its histological characters. Both the sections stained with eosin-hæmatoxylin, and with aniline dyes, and those simply hardened in the osmium-bichromate mixture, without further process of staining, give a separation of the organ into three portions; all of which are microscopically dissimilar in appearance. There is first an outer lamina of slightly irregular ependymal cells (*a*, fig. 2), three or four deep, arranged after the manner of the cuticular epithelium, separated into divisions by thin processes extending from the fibres of the surrounding capsule and terminating at a definite line, where a separation from the more internally lying elements occurs; then follows a more internal zone of varying depth containing epithelial cells of a secretory type, which in places along the posterior and inferior border of the lobe, as well as occasionally in the more central regions, are arranged into distinct closed acini, lined with a low variety of cylindrical epithelial cell, and often hold in their lumen collections of a colloid substance. Occasionally, several of the acini are seen coalesced, forming cavities of small size. The follicles vary very greatly in numbers in the several sections, but are always more numerous along the posterior border than elsewhere, though they may be present quite up to the mid-region of the lobe when invaginations of the outer epithelial layer occur. Intermingled with the acini are numbers of smaller cells, not so distinctly epithelial in appearance, which become predominant in the central and more anterior regions.

The alveoli are separated by trabeculæ of connective tissue, bearing blood-vessels, which are not of such large dimensions as are found in the anterior lobe.

This secretory region gradually merges into a central region of small rounded and polygonal cells, separated by extensive connective tissue partitions, carrying blood-vessels, and scattered widely among these cells are others of larger dimensions. Among the scattered cells may be distinguished some of spindle form, others of pear or rounded shape, and still others of very irregular form, with their borders ill defined; all having a finely granular appearance, with here and there larger granules scattered among them, that are tinged by osmic acid a blackish colour. The nuclei of the cells, when they can be determined, are rounded, have a nucleus and adnuclear particles, and are moderately large in size. Along the anterior border of the lobe, the epithelial elements, except the outer ependymal row, retreat a little in prominence, especially along the anterior margin adjacent to the region, where in the lower orders of animal life the infundibular duct is present (now represented in the dog, only by blood-vessels and connective tissue bundles), they become segregated into groups of mainly oval and pear-shaped cells, separated by a fine stroma, with small nuclei here and there in it, and are now easily recognised as nerve cells. A certain number of the spindle cells are very long as well as broad, and probably correspond to those described by Krause, as spindle cells of uncertain function, but are undoubtedly nerve cells.

It has been shown by Andriezen,[1] that the pituitary in *amphioxus* and *amocœtes*, is of three-fold structure, namely, a sub-neural glandular organ; a duct lined by ciliated epithelium, which affords a communication between the buccal and neural cavities; and a group of nerve cells, around and at the back of the upper opening, where the duct widens into the ventricular cavity. He has further shown that particles of carmine, suspended in the water surrounding the animals, will be taken up with the water passing through the infundibular duct, and carried by ciliary action into the ventricle, and thence into the central canal of the cord, finally the particles of carmine may be traced right up to the free end of the canal, where the spinal cord opens into the exterior by the blastopore; therefore, it is made manifest, that the infundibular duct carries a stream of oxygen-bearing water, for the nutrition of the tissues, and the carrying off of their effete products.

It is quite curious to find essentially the same structures preserved in as high a vertebrate as the dog, and descending to so low a zoological order as amphioxus, though as Müller remarks, the pituitary is practically the same from *myxine* to man. The epithelial structures surrounding the proper nervous portion of the pituitary, are hardly to be accounted for, except on the supposition that they originally enclosed a group of nerve cells, whose processes, probably, through a connection with peculiar neuro-epithelial cells, had the function of testing the water entering the infundibular canal (Herdman, Andriezen), and that after this function had become lost in the gradual ascent in animal life, these cells became metamorphosed into cells having a secretory function, in part, homologous to those of the anterior lobe, and probably originated from an offshoot of the same buccal epithelium that formed the substance of the anterior lobe. The nerve cells of the ganglion likewise underwent partial changes, a group of them remaining near their former situation, and retaining somewhat of their old characteristics, while the deeper ones became more scattered, through the infiltration of new cellular elements between them, even to the extent of producing a partial loss of function, and consequent prevention of their further and higher organization.

---

[1] Andriezen, *British Medical Journal*, Jan., 1894.

We have been so fortunate in a number of specimens as to obtain good staining of the nerves and neuroglia elements of the nervous lobe of the pituitary, but unfortunately the nerve and neuroglia cells, and the nerve fibres, do not stain well in the same sections, the fibres being better influenced by the plain Golgi, the neuroglia and nerve cells coming out with greater clearness and frequency in the picric acid preparations, hence, the connections between the two portions of the nervous structures are in some measure difficult to ascertain.

The Supporting Tissues.—At the outermost margin of the body, there are seen extending from the thin capsule surrounding it, numbers of rather thick varicose threads, all unbranched, and invariably ending when their terminations can be discovered, in a ball-shaped figure, at a definite line in the substance of the body, usually at the inward ending of the first layer of epithelial cells, at the line of separation from the more centrally situated elements. These knobby threads strongly resemble the ependymal glia cells of embryonic life, and possibly may be related to them, but as their basal end is shrouded in a blackened aggregation of cellular masses, their histological origin must remain a matter of some uncertainty. Throughout the remaining portions of the lobe, connective tissue trabeculæ, are everywhere met with, separating the cellular elements, and bearing the blood-vessels.

In the posterior two-thirds of the body, glia cells are not stained, but as we approach the anterior edge, that is in all portions of the anterior third of the lobe, where the nerve cells are thickly strewn, we find moderately numerous neuroglia cells of different types, principally a mossy cell of varied form (fig. 3), lying between the groups of nerve cells; and the usual spider element, with its single very prominent tentacle (fig. 4), but their bodies are a little different from those seen in the purely cerebral tissues, being much larger in proportion to the extent of their tentacles. In the superior portion of the organ, adjacent to the infundibulum, these spider cells are in moderate numbers, to the exclusion of every other variety, though nowhere in the substance of the lobe are very numerous glia cells of either type stained.

The Nerve Cells.—It is only here and there through the anterior two-thirds of the lobe, that the silver stain affords examples of the varieties of nerve cells of the body. At the extreme base a small group of cells may quite constantly be seen stained (*g*. fig 2), but this is the only example of groups of nerve cells stained by the silver salt, elsewhere they are only tinged in singles and in collections of twos and threes. In some respects this defect in the reception of the silver salt is a drawback, in others it is a decided advantage, as the full figure of the tinged cell stands prominently forth among the unstained elements, with their exceedingly beautiful and massive neurodendrites.[1] It is hardly to be supposed that any varieties of the nerve cells have escaped impregnation at some time in all of our sections, as the isolated cells are pretty thoroughly scattered among the regions occupied in the unstained sections, by visible nerve cells. All the ganglionic elements of the gland may be considered as belonging to the multipolar type, no exception to the rule being determined by the method of staining.

For the sake of distinction the nerve cells may be divided into two principal classes, cells with one neuraxôn, and cells with two or more neuraxôns.[1] These varieties may be further subdivided into large irregularly pyramidal and oval cells having large numbers of neurodendrites and a single neuraxôn (figs. 5 and 6); medium-sized multipolar cells of very irregular shape with many dendrites, among which may be distinguished one very prominent one directed towards the upper and anterior margin of the lobe (fig. 7); irregular pyramidal cells with the same characteristics (fig. 8); flasked-shaped cells of about the same size with numerous processes, from which come off at different points on the dendrites, two or more axis-cylinder processes (fig. 9); very small pyramidal and flasked-shaped cells, having an enormous length for their apical processes (figs. 10 and 11) ; rounded cells of equally small dimensions with very strong processes, among which may be distinguished a single axis-cylinder extension (fig. 12) ; lastly, cells of very small size, situated along the extreme upper and inner edge of the lobe, with an apical tuft of smooth protoplasmic processes, and an axis-cylinder extending downward toward the centre of the lobe (fig. 13).

---

[1] I have throughout this article used the word neurodendrites, and dendritic and protoplasmic extensions or processes, also neuraxôn and axis cylinder, as synonymous.

(1) The first variety (figs. 5 and 6), the largest pyramidal and oval cells, are possessed of many and strong dendrites, which come off from the thickened points at the extremities, and along the margin of the cellular body, and gradually break up into finer and finer ramifications, which eventually terminate in filaments of feathery tenuity, giving the cell a very beautiful appearance. The axis-cylinder processes of these cells, which is always one to each cell, throw off very few collaterals near the cell body, but later break up into numerous branches, some of which terminate upon other cells, while the main thread continues upward, toward the upper periphery, to end in a very complex network among the epithelial cells along the border of the lobe. These larger multipolar cells are principally found among groups of unstained cells along the anterior and inferior margin of the lobe.

(2) The medium-sized pyramidal and irregularly oval cells (figs. 7 and 8), are found in numerous locations, both along the borders of the organ, and in the more central regions, quite as far back as the nerve cells extend. Their bodies are usually well stained, but the dendrites are quite short, with the exception of one very long one, which may arise from either the apex or side of the cell. These dendrites exhibit none of the extensive ramifications and arborizations of the larger variety, but on the contrary are often distinguished by the presence of numerous hairy processes, extending but a little distance from the ramus. These hair-like processes may also come off from the margin of the cellular body itself (fig. 8). Clubbed endings to the dendrites

are also more frequent than with the larger variety. Always among the dendrites may be distinguished a single one that does not diminish in calibre as rapidly as the others, and extends for a long distance from the cell, with a general direction upwards and forwards. The neuraxôns are not very easy to distinguish from the other processes, and appear to originate by a gradual diminution of the calibre of the process. Each cell seems to possess only a single neuraxôn.

(3) The flask-shaped cells (fig. 9) are to be found over a wide territory, and while covering a considerable space with their branches, never show the fine feathery arrangement of the largest cells, but finally pass by a gradual diminution of their calibre into finer and finer neurodendrites, ending free among the tissues, frequently with a small knob-like arrangement. From two to four neuraxôns have been found coming off from this variety of cell, and though it is difficult to trace them for any great distance, they would appear to break up into a fine meshwork which surrounds unstained cells.

(4) The small flask-shaped and pyramidal cells (figs. 10 and 11) are moderately numerous, and are found chiefly in the central regions of the organ. They do not show many basal, and these are only stained for short distances, but on the contrary they have exceedingly strong apical processes, which may run almost through the long diameter of the body, giving off lateral processes as they extend upwards, and these processes have at times a tendency to form a complex brush-like figure at their terminations. No axis cylinder process is to be made out, coming off from these small cells, but the appearance of some of the basal processes would indicate that the origin of the neuraxôn was from that extremity. The singular endings of some of the apical processes would indicate a peculiar function for these cells.

(5) Our next variety is an interesting type of cell, of which the drawing hardly gives a complete idea. The body is small and of a rounded form, from which strong dendrites come off, and sub-dividing, cover a large space around the body of the cell. These ramifications have exceedingly numerous thorny and knotty extensions from them, giving the dendrites a prickly appearance. The axis-cylinder

process comes off at some variable point from one of the thickest arms, and is fine, and shows collaterals here and there, though they are not very numerous. This cell is only stained near the upper border of the body, and the axis-cylinders soon disappear among the thick-set arrangement of the nerve fibres found in this region. It is very probable that this cell belongs to the intermediary (second type of Golgi) type, though, as already mentioned, but one neuraxôn is to be found belonging to it. This cell is peculiar to the region, and to the lobe, so far as I have been able to determine, not having met with a similar variety of ganglionic body in any other portion of the central or peripheral nervous system.

(6) The last variety is a cell of rather smaller size than than those of sub-class four, and is quite numerous along the upper and anterior border of the lobe, and is distinguished by a strong tuft of fine wavy prolongations from the upper apical portion, which extends almost, but not quite, to the inner free margin (fig. 13, also fig. 2), where they are intermingled with the network of varicose nerve fibres, that are so thickly set in this region, along the space formerly occupied by the infundibular duct. The basal processes are inconsiderable in numbers, at the most two or three, and do not give off lateral branches over considerable distances. They are directed downward, and finally intermingle with, and are lost among, the haze of nerve fibres in this region.

The chief protoplasmic extensions of all the larger and medium-sized nerve cells are distinguished either by their extreme length, or by the extent of territory over which they spread. The extensions themselves are either knotty, prickly, or have a feathery arborescence developed upon their branches, which is simply the breaking up of the finest protoplasmic prolongations, into exceedingly numerous fine short ramuscules.

One of the peculiarities of the cells of the lobe is the development at the final ramifications of the dendrites, of complex tufts of various forms, which, under low powers, show only as irregularly blackened objects, lying among the thickly set protoplasmic extensions. These tufted objects are frequently seen among the processes of the lowermost group of cells (fig. 2, also fig. 14, in which one of the developments more highly enlarged is reproduced). Into it, two varicose fine processes are seen entering, which sub-divide and form figures of peculiar shape, with numerous irregularly blackened points and knotty knobbed endings, extending from and scattered among them. Tufted developments of more simple form are occasionally seen among the extensions of the larger cells, situated in the mid-region of the gland.

The ramifications of the smaller cells, all of which, with one exception, are of moderate length, may be divided into four groups: (1) Those that branch at numerous rectangular and obliquely angled points, but whose finer dendrites do not sub-divide to any extent (fig. 9); (2) cells whose processes sub-divide at numerous rectangular and obtuse angles, and are covered with a multitude of feathery and spicular projections (fig. 12); and (3) cells of irregularly oval form, with tufts of thin non-varicose processes; lastly, cells with very long, not frequently divided apical processes, and short basal ones (figs. 10 and 11). None of these cells show other than a usual development of the dendrites, with the exception of the last variety, where we again find tufted endings, though of more simple form than those appertaining to the larger pyramidal and irregular cells. These tufts are scattered widely in the central regions of the lobe, and as certain of the nerve fibres, as will presently be seen, also develop bushy end-figures, it is sometimes difficult to distinguish between termination of axis-cylinder, and termination of dendritic extension.

The Neuraxôns.—The majority of the axis-cylinder extensions of all the numerous varieties of cells belonging to the organ, are by their finer calibre, and the constant presence of small characteristic varicosities and thickenings, readily distinguishable from the dendrons; but in certain localities, notably in the mid-region of the lobe where the finer lateral extensions of the pyramidal and flask-shaped cells are moderately numerous, we find ourselves between the horns of a dilemma to decide which is axis-cylinder or nerve fibre and which is protoplasmic extension, and it is only by following them to their terminations, when such may happily be found, that we are definitely able to determine the difference.

The axis-cylinder extensions of all the cells in the inferior portion of the lobe turn upwards; the direction of those belonging to the smaller pyramidal and flask-shaped cells, I have not been able to determine. Those belonging to the larger proportion of the smaller cells of the superior border turn upwards, and intermingle with the marginal fibre network, and but a few belonging to the tufted cells of the higher regions probably turn downwards toward the central regions, and are soon lost in the maze of fibres.

All through the middle and anterior portions of the lobe, the intermingling of the neuraxôns and neurodendrites forms an exceedingly complex arrangement, in which it is difficult to pick out the individual fibre belonging to one particular cell, and trace it for any considerable distance. All the axis-cylinder processes, and the long dendrites, have a general tendency upwards and forwards, both dendrites and neuraxôns branching as they proceed onward, but all traces of the dendrites of the inferior and median cells of the lobe are lost some little distance below the superior edge, and then the neurons only are intermingled with the extensions of the smaller superficial cells, passing them, however, before the border is finally reached, where they spread out into a most extensive felt-work of fine varicose fibres, still retaining something of their previous longitudinal arrangement from the threads of the uptending fibres being coarser than the lateral and intermingling branches. It is doubtful whether any of these fibres pass beyond the limit of the lobe into the infundibulum, our sections giving no evidence of such an arrangement; while, on the other hand, the nerve fibres accompanying the larger arteries are sometimes distinctly seen coming from the infundibular tract into the body of the posterior lobe of the gland, and ramifying through it. It is probable, too, that some portion of the nerve threads in the posterior secretory portion of the lobe are also derived from these vascular filaments, but connections between the fibres of the vascular supply and the nerve cells of the organ we have never been able to observe.

A small number of the nerve fibres originating in the lobe have thickenings of some size in their course, and more rarely a pear-shaped diverticulum of small diameter is seen extending from a fibre, the appearance being similar to certain cells of the spinal ganglia having T-shaped extensions, only they are much smaller.

In the central regions of the lobe certain rounded or oval bodies are observed (*v.v.*, fig. 2), which in the chrome silver preparations have a likeness to closed follicles, but have no visible structure, and only appear as oval bodies of a yellowish colour with a sharply marked line of differentiation from the surrounding structures. A careful search through a number of eosin-hematoxylin preparations failed to reveal any similar rounded bodies in the central regions of the lobe, though in several instances the follicles pressed far inward. Around these bodies the axis-cylinder processes coil with an intricate arrangement, the main fibres in their upward course diverging around them, but from the same fibres arise others, that, after passing to the bodies having the outline of follicles, develop upon themselves very extraordinary figures (15 and 16, also fig. 2, *v.v.*). These fibres twist and coil (fig. 16) upon themselves, develop irregular thickenings, the most common being an irregular, comb-like figure, with knobs set on the end of the blades. There is also a tufted form, and sometimes only a development of intricate knobs is observed, extending from a blackened mass, having a nerve fibre passing into it.

With the exception of the ones present among these peculiar endings, there do not appear to be anastomoses among any of the fibres of the lobe; they branch repeatedly, cross each other, their lateral and even main stems develop arborizations around other cells, especially clearly seen around those that are not stained, but nowhere, even in the situations along the anterior border where the nerve threads are sufficiently dense to form a feltwork, do they ever anastomose.

In the posterior third of the lobe, among the epithelial structures, the nerve arrangement, owing to the greater though comparative rarity of the stained elements, is seen with exceeding clearness and distinctness. Here varicose fibres come in numbers from the median portion of the organ, branching repeatedly as they wander through the tissues, becoming finer and finer in calibre, and finally end with free minute branches apparently without end-knobs, though this may be a defect in the staining.

Numbers of these nerve threads cross the line separating the follicles (fig. 2) from the outer layers of epithelial cells, and form outspread arborizational figures of considerable extent. Anastomoses between these filaments do not occur. Here also no end-knobs are to be found, though the staining of the fibres is very clear.

The silver method shows the pituitary gland to have retained, in one of the highest orders of vertebrates, its double *rôle* of secretory and nervous function, intact; the former perhaps modified, the latter, the original special sense organ, probably simply lying quiescent, not atrophied, and only changed in so far as to admit of a slightly different arrangement of its constituent elements.

It is exceedingly difficult to interpret the meaning of the peculiar comb-like figures and nerve tufts upon the central nerve endings. If the rounded bodies are follicles, their nerve endings are certainly very dissimilar to the endings upon the acini of the salivary or thyroid glands, or even the adrenal capsules; very much more strongly do they resemble the endings of the mitral cells of the olfactory bulbs, the so-called olfactory glomeruli, and it is more plausible to refer them to some organ of special sense existing fully developed in the lower forms of animal life, than to endings upon secretory epithelium.

II.—The Infundibular Region.

Those portions of the inter-brain of embryonic life, represented in the adult by the floor of the middle ventricle, may, for the purposes of this study, be divided into three tracts; the regions anterior and posterior to the central opening of the ventricle, and the funnel-shaped extension downward, the infundibulum proper: the component parts being united by the rounded walls of the infundibulum, and fused together at its base without a distinct line of differentiation between them.

The Infundibulum.—In eosin-hæmatoxylin preparations of this lowermost portion of the walls of the third ventricle, we find a moderately thin lamina of fibrillated tissue containing a multitude of nuclei of rounded or angular form, a few nerve cells of pear or rounded shape, and a number of large blood-vessels; the whole tract being divided, somewhat unequally, into two parts, by differences in the reception of the staining agents by the tissues. The outer half of the lamina has fewer nuclei, and a finer as well as more complicated arrangement of the glia fibrils than the inner, where the component fibrillæ are coarser and less complicated, and the nuclei more abundant. The central point of the most inferior region of the infundibulum is united to the apex of the posterior lobe of the hypophysis by a mass of blood-vessels and fine fibrillated tissue, with considerable numbers of nuclei among the bundles; the whole arrangement of the structures of the infundibulum being here altered. Elsewhere it shows no break in the described arrangement, the line of differentiation between hypophysis and infundibulum being sharply drawn, a layer of coarse connective tissue bundles being placed between, and separating the glandular and other structures of the pituitary from the tissues of the infundibulum. The staining of the infundibular tissues by chrome-silver, shows an exceptionally complicated mass of interlacing glia elements of a distinctly embryonal type, which is so dense, that in fully impregnated sections, it is impossible to see more than a complicated mass of rods, without differentiation into cellular forms.

In less thoroughly stained specimens we may readily distinguish two principal types of neuroglia cells (1 and 2, fig. 17) and several sub-varieties. In all portions of the organ up to its fusion with the tuber cinerium, we find glia cells passing from the internal edge of the ventricular portion of the lamina to the extreme outer margin of the infundibulum, resembling in every respect the earliest ependymal glia cells of embryonal life, and corresponding to those arising from the ependyma of the lateral ventricles and penetrating through the cerebral tissues. At the edge of the ventricular cavity we see small pyramidal and elongated cell-like bodies (1, 1, fig. 17) with a slight projection into the cavity of the ventricle, each cell giving origin to a single thick stem, often slightly roughened, which, after reaching as a single twig the middle or even superior third of the lamina, breaks up into several branches, bearing upon themselves small numbers of rounded thickenings. Finally these branches end at the epi-infundibular limit in large rounded or flattened knobs, the latter form often having the aspect of being cup-shaped at their outer extremities. This variety of cell is sometimes stained to the exclusion of every other ependymal form, and then occupies the whole infundibular tract to the beginning of the substance of the tuber cinerium.

The first sub-variety (2, fig. 17) consists of straight, very rough fibres, arising from the edge of the ventricle, only here and there showing cell-like forms at their bases, and running almost to the edge of the outer limit before ramifying. There they sub-divide into a few rather fine branches, only occasionally having knob-shaped terminations upon their extremities. Sub-variety two (3, fig. 17) comprises exceedingly coarse fibres arising from ependymal cells, often of quite irregular form, and almost immediately break up into ramifications after their origin from the cellular protoplasm, finally terminating beneath the outer limit. When the border is reached the knob-like ending is always apparent, consequently the other form, where they end below the margin without a swelling, is probably an imperfection in the staining. These cells are very numerous, especially in the lateral portions of the body, and supplant the primary form (type I) of ependymal cells. Sub-class three (4, 4, fig. 17) is only stained in the outer half of the infundibular lamina. Their stems are sometimes very thick, and sometimes very thin, but always at a certain point they develop an irregular or triangular thickening, out of which sprouts a variable number of fine threads to make a brush-like figure. The threads, when they reach the periphery, are so fine and closely set together that they look like a blackened spot along the outer margin. None of the branches of the ependymal cells ever anastomose.

The second, and only other type of glia cell (12, fig. 17) that is stained by the silver salt, is a rounded cell of quite large dimensions, having a multitude of exceedingly delicate varicose processes extending from the body, that never branch or anastomose with one another, and give to the cell a burr-like appearance. This type of cell is but sparingly stained, and is present in all portions of the body, most frequently within the inner half, though occasionally they may be found quite close to the outer limit.

The Nerve Elements.—Both the nerve cells and fibres stained by the chrome-silver are quite sparse in numbers when compared with the neuroglia elements. In the unstained osmic acid preparations but a few rounded and pear-shaped cells (5, 5) are to be discovered. The cells of nervous function tinged black in the silver preparations resolve themselves into the usual varieties, cells with one neuraxôn, and cells with several neuraxôns, all being multipolar.

These types may be sub-divided into four classes: (1) Large multipolar cells with fairly numerous strong processes (6, fig. 17), among which may be distinguished a single one which extends, without branching in a circular direction some considerable distance from the cellular body, and then develops knots and triangular irregularities (10, fig. 17) in its course, from which arise rather thick, right-angled rami, that end, without again branching, at the periphery, in a globular enlargement. The main filament continues in its circular course, and finally disappears before arriving at any definite termination. (2) Smaller pyramidal cells (7, 7, fig. 17) also situated in the long diameter of the body, which gives off one or two apical processes following the direction of the long extension of class 1, though they do not develop lateral processes, except in occasional instances, nor is their protoplasm stained to any definite ending. (3) Cells of pyramidal form (8, 8, fig. 17) but of smaller dimensions, lying transversely or obliquely to the surface, that have an axis-cylinder process coursing among the sparsely stained longitudinal fibres, and knobbed-endings to their pyramidal processes attached to the outer limit of the infundibulum. (4) Small cells of pyramidal form (9, fig. 17) with a considerable number of basal processes, and a large number of fine, smooth, apical ones, from which arise a number of axis-cylinder extensions, coursing circularly through the long direction of the body, where they are eventually lost.

Nerve Fibres.—The irregular protoplasmic extensions of a number of the largest pyramidal cells found in every portion of the infundibulum, we think should be looked upon as axis-cylinder extensions of probably strictly local function, though from their irregular appearance and the development of triangular thickenings it is improbable that they are provided with a myeline sheath. The other neuraxôns, all of which belong to common forms of nerve fibres, are sparingly arranged after a circular fashion entirely in the mid-regions of the body. They are quite fine, possess numerous varicosities, and have the same general appearance as the finer fibres of the cerebrum when stained by chrome-silver. No fibres of nervous origin, apparently, pass to and fro, between the posterior lobe of the hypophysis and the infundibulum besides a few vascular nerves.

In well stained sections, a few thin nerve fibres (11, 11, fig. 17), may always be seen passing beyond the limits proper of the infundibulum into the gray substance of the lateral wall of the third ventricle, along which they ascend for some distance and are finally lost in the haze of nerve and ependymal glia fibrillæ about two millimetres above the opening of the ventricle into the infundibular cavity.

III.—The Neuroglia of the Ventricular Walls.

As we approach the opening of the infundibular cavity into that of the third ventricle, we find the ependymal cells assume a different form from those already described. The cellular mass at the base is now very small (fig. 17, No. 14), the cells closely set together, so closely indeed that they frequently appear to be fused into a single mass, out of which extend long, very infrequently branched rays, studded here and there with bead-like thickenings. These rays extend through the gray tissues almost incredible distances, passing in the direction of the basal ganglia. There is only a single process to each cellular body.

Proceeding upwards along the borders of the walls of the ventricle, we immediately come upon another and most beautiful variety of ependymal neuroglia cell (fig. 17, Nos. 15, 15). This cell is somewhat different in form from any of the familiar types that have been depicted by Retzius, Kolliker, Cajal, and others. The body is fairly large, of a rounded or flattened form, and is situated along the margin of the ventricular cavity, and very often sends out a short spiked process into the ventricle. Out of the body very numerous lateral rays proceed, extending along the margin of the ventricle for considerable distances. From that portion of the cell body directed towards the cerebral substance, grows a main process which is primarily single, though later it may divide into two or even three main branches. This vertical process has projecting from its sides large numbers of rectangular lateral tentacles, some quite short, others of medium length, similar to those passing from the body of the cell, and taking the same general direction. Some of these extensions are thicker and shorter than the others, and have knotty and prickly projections from their margins, while others are longer and have the prickles, but no knotty thickenings. Subdivisions of the lateral processes are somewhat rare, but do occur. Out of the uppermost portion of these strong vertical processes, we can almost always find a long, smooth, hair-like prolongation, which shortly bends downward toward the floor of the brain, where it meets the up-tending fibres of the sustentacular glia cells along the inferior margin of the floor, as well as the rays from the last described variety of cell, and mingling with them, is soon no longer distinguishable in the dense meshwork; though it is probable from the appearance of a few isolated examples that they all end in a ball-swelling against the pial limit. The general effect given by these ventricular cells reminds one strongly of a lateral view of some of the varieties of conifers, hence we have named them the *fir-tree* ependymal cells.

These very beautiful bodies are thickly set along the margin of the ventricle, and are found in perfection up to a height of several millimetres above the debouchment of the ventricle into the infundibular cavity, above which they become less frequent, and are dislocated from the immediate border of the ventricle, appear to become stunted and lose their long, smooth prolongation. Some of the examples of these cells are very large, others quite small, but together they fill up the whole space along the edge of the ventricle in this region to the exclusion of every other form of neuroglia cell. There are no transitions between these neuroglia cells and the more common varieties in their neighbourhood, and they are stained in no other location than along the immediate border of the ventricle.

A little outward from the ventricular edge we begin to meet with cells approximating the short-rayed Golgi cells in shape, but their bodies are of rather larger size, the tentacles coarse, not reaching to the ventricular border (fig. 17, No. 16), and it is only when we approach a region about 3 mm. from the pial border of the basis cerebri, and some considerable distance from the ventricle, that the distinctly glia cells of the long-rayed type first make their appearance, and are then very numerous and closely placed together, their rays interlacing in all directions. In specimens in which the neuroglia is fully stained, the meshwork formed by the prolongations of these cells is so dense, even without the admixture of nerve filaments, that under low powers this entire portion of the section is stained almost a solid black.

Returning now to the floor of the brain lateral to the ventricular cavity we again see the shapes of the neuroglia cells change completely. The embryonal types have everywhere departed and in their place, ranged along the inferior edge of the brain floor and from thirty to sixty mikra above it, are stout-bodied cells of irregular shape (fig. 17, Nos. 17, 17), with processes extending in all directions, interlacing with each other and forming a grill-work, but never anastomosing. Two chief types of cells may here be differentiated. The first (Nos. 17, 17) are cells with irregular bodies and with a vast number of rays extending from all portions of the cellular protoplasm, but with several thicker processes extending upward into the cerebral substance, and a number, often very considerable, of shorter ones passing to the pial limit, where they end with ball-like knobs. The lateral rays from these cells are numerous and strong, and help largely to form the interlacing network along the margin of the brain floor.

The second form (fig. 18) is an extremely angular cell, having quantities of hair-like projections from the cellular body and basal portions of the thicker processes. The extensions themselves are not so numerous as those of the former type, and are only notable by arising from the body by thick junctures and then gradually diminishing in calibre. Among the prolongations one may always be distinguished by its stout appearance, which is directed upward toward the central regions of the brain, there to terminate either by a gradual process of attenuation, or by ending in a cone-shaped figure.

Along the borders of the optic nerves and among their fibres the neuroglia cells return to the long-rayed Golgi type, but their processes are of far thicker calibre and coarsely but infrequently beaded, and visible among the nerve structures much longer distances than the ordinary long-rayed cells, the two forms being drawn for contrast in figures 21 and 22.

Sixty to eighty mikra above the inferior margin of the brain we first find interesting transition forms between the cells already described and others of probably later development. In fig. 12 of the main figure we find an illustration of an irregular cell with quite large body and numberless rays of considerable length extending in all directions from it. These processes are further subdivided into longer and shorter rays of non-branching character, and stouter short rays with a few short branches, both processes and their branches being covered with minute hair-like projections, giving the arms a shaggy appearance. On the opposite side of the drawing a similar cell is depicted (fig. 17, No. 13) of the same general character, but with few smooth processes of any length, these often bearing at their terminations globular figures. The other extensions of the cell are short and thick and are covered with the fuzzy hairs.

Very numerous forms of similar cells are present all through this region, among them (fig. 19) a cell with pyramidal body, feathery prolongations, and numerous hairy projections from the body and basal portions of the thicker extensions, the type resembling that last described, only being more delicate. These bodies finally give place, higher up in the tissues, to the long-rayed Golgi cells, intermingled with a few of the short-armed variety.

It is at times almost impossible to distinguish between these hairy cells and some of the smaller varieties of nerve cells situated in the circumjacent regions. On the left side of the drawing (No. 20), the same hairy appearance of the body and main arms can be noted, also the globular endings to the processes directed downwards, and when the neuraxôn of the nerve cell is not stained, it is utterly impossible to differentiate between these varieties of nerve and similar shaped neuroglia cells.

The last variety of glia cell (fig. 17, Nos. 21, 21), that is present along the borders of the ventricle, though never nearer than 50 to 80 μ. from it, and never very near to the base of the brain, is a cell with a body of variable size as well as irregular shape. Extending from the protoplasmic body are a variable number of stout prolongations, and these extensions, as well as the body of the cell itself, are covered by a vast number of thorny and knobbed processes, generally of short extent, giving to the entire cellular body a very shaggy appearance, comparable to a mass of coarse moss with a central more dense portion, hence the name of mossy cell.

Very similar cells are depicted by Retzius[1] from a preparation of a 26 cm. long human fœtus, occurring within the gray columns of the spinal cord; also somewhat similar figures are given by Andriezen[2] from the cortex of the brain, under the name of protoplasmic glia cells. Some of these cells are of very large size, and stretch between the nerve bodies and fibres, covering an immense territory. Nearly all of them have one or two longer extensions, that eventually lose their mossy appearance, and run as almost straight fibres through considerable areas of the brain substance.

---

[1] *Biol. Unters. N.F.V.*, 1893.

[2] *Brit. Med. Jour.*, January, 1894.

I was at first inclined to think that this mossy appearance was increased by a deposit of silver precipitate in coarse form upon very slight lateral projections from the main arms, but afterwards found numerous equally shaggy bodies in sections entirely free from every trace of deposit.

All the different described forms of neuroglia cells, with the exception of those belonging to the embryonal types, are in close relation to the blood-vessels of their vicinity, the connection being made by the application of their globular endings to the hyaline sheath of the vessel..

Two of the most distinguished, as well as recent writers on the histology of the central nervous system, have stated that, in their opinion, the embryonal supporting substance of the brain and spinal cord—the ependymal neuroglia—almost entirely atrophies and disappears in the adult mammal. To be more exact, Cajal[1] states that in birds and mammals "the epithelial neuroglia retains its embryonal type only in two organs, the olfactory mucous membrane and the retina;" while Kölliker,[2] speaking of his attempts to obtain a successful staining of the ependymal cells of the spinal cord in full grown animals, adds, after reciting his want of success: "I presume, therefore, that the ependymal fibres are only present in the adult in a very stunted condition, and it is not to be supposed that they run to the outer surface of the cord."

After reading these statements it was something of a surprise to find the above described beautiful specimens of several types of ependymal neuroglia extending from all portions of the middle and inferior regions of the cavity of the third ventricle, and reaching to the periphery, all portions, bodies, branches, tentacles, and sub-pial endings, being readily distinguishable.

The region examined is therefore very interesting, not only from the great variety of neuroglia cells that may be seen within a very limited area, but from the fact that varieties of the ependymal neuroglia cells, previously supposed to have entirely disappeared from the central nervous system in the adult mammal, are found present in perfect condition in the brain of a very high order of animal, and are not confined, as has previously been supposed, to those of adult reptiles, amphibia, and fishes.

---

[1] Cajal, "Neue Darstellung v. hist. Bau d. Centralnervensysts." *Arch. f. Anat. u. Phys.*, 1893.

[2] Kölliker, *Gewebelehre*, *6te. Auflage*, 1893.

IV.—The Nerve Elements of the Tuber Cinerium

The region lateral and inferior to the descending limb of the cavity of the ventricle, and its anterior and posterior *cul-de-sacs*, presents no important differences between the nerve elements situated in the several departments, and accordingly they will be considered together.

Above the region comprising the floor of the tuber cinerium, containing the layer of interlacing neuroglia cells, lies a zone having comparatively few cellular elements, either stained or unstained, in it, mainly comprising small pyramidal and angular cells with rough processes, intermingling with the filaments of the glia cells. The major part of this region is filled with the dendrites of higher located cells, intermingled with fine branching axis-cylinder processes, the extensions mainly of intermediary cells, of adjacent but over-lying regions.

Situated immediately over the sparsely apportioned pyramidal and irregular cells, and mingling among their upper members, are well stained spindle and angular cells of a number of types and sizes, and above these are pyramidal, globular, quadrate and angular cells intermixed with small numbers of the spindle-shaped cells, this arrangement extending to the limits of our sections. None of the stained cells of this region are large, with the exception of a few pyramidal cells of the upper limit. No large nerve fasciculi are to be seen in the region, with the sole exception of a round bundle lying horizontally and laterally to the ventricular opening, which is better developed in the mouse than in the dog, but numerous small bundles of medullated fibres penetrate from overlying regions, and into them the greater portion of the axis-cylinder processes of the projection cells appear to pass.

The Spindle-shaped Cells of the Middle and Lower Zones. —These nerve bodies are the most frequently seen cellular forms stained in the region immediately super-imposed on what we may call the barren zone, and though almost every cell departs somewhat in shape from its neighbour, three principal forms may be described. All these cells lie either vertically or slightly obliquely to the brain surface.

(1) Spindle-shaped cells with short bodies (fig. 22) having regular outlines, and strong processes arising from either extremity, perhaps immediately sub-dividing into two or more divisions which have numerous short lateral spiculæ and knob-ended projections from the sides. The processes directed upward are thick, and, so far as can be determined, do not sub-divide to any extent, though some of them can be traced over long distances from the cell body. There is commonly only one strong process directed downward, which passes, giving off short branchlets on the way, down to the barren zone, there to end, after the development of many beads upon it in a conical termination. Some of these extensions may be traced nearly up to the pial limit. Other downward directed processes are shorter, and terminate in a rather peculiar thickening, from which is thrown off short right-angled fuzzy branches (fig. 22), but among them can sometimes be distinguished a very much finer extension, that turns upwards and is soon lost in the haze of nerve fibrillæ surrounding the cells. The main axis-cylinder process, if so we may speak of a cell that always has several neuraxôns arising from some point of the protoplasmic extensions, is directed upward, waving through the tissues as it proceeds onward, and soon sends out a few collaterals, some directed laterally toward small cells of the locality, while others turn downward toward the lowermost cells, where they divide repeatedly and form wide ramifications with a finely varicose character to the threads (fig. 29), finally encircling the nerve cells and ending upon them. The further course of the uptending neuraxôn is a matter of some uncertainty; a few of them appear to join small nerve bundles and proceed to higher

regions, but the majority are soon lost by a change of course and disappear. The other neuraxôns belonging to the cell simply divide between surrounding cellular forms.

The second class of the spindle cells comprises bodies having an irregular outline, but the chief distinction is in the arrangement of the protoplasmic prolongations. Near the cell body they are slightly roughened, a disposition which increases as the distance from the central point lengthens, and when the extremity is reached (fig. 23), they sub-divide into a large number of short branches thickly studded with beads, and forming striking objects under high powers. These cells have several neuraxôns, usually directed laterally, but they cannot be traced any great distance among the tissues.

The third variety of spindle cell comprises very small cells, rather long, but of scarcely greater diameter than their immediate neurodendrites. The extensions branch considerably, and extend over considerable areas, but are not so frequently sub-divided or varicose as the last type. Their neuraxôns tend upwards.

In the same territory as the spindle cells we find fairly numerous cells of angular form (fig. 24), with a single axis-cylinder process extending upwards and sending off collaterals at frequent intervals, which finally enter some of the small bundles of medullated fibres passing beyond the limits of the section. The dendrites of these cells hardly branch at all, but are ofttimes very long and always roughened by short spiculæ from the sides, the cell body remaining smooth in outline.

In the neighbourhood are very similarly shaped cells with two or more neuraxôns, and it is very possible that all of these cells are of the poly-neuric type. An out-spread axis-cylinder prolongation of one of these cells is drawn in fig. 30, where the single nerve thread entering the superior portion divides and re-divides, forming very intricate figures.

Intermingled with the cells of angular form are medium-sized triangular and pyramidal bodies with stout, roughened processes (fig. 25), coming off at the angles of the cellular body. These cells lie with their long processes directed downward, upward, or laterally, with reference to the surface of the brain. Some of the terminations are of very great length, passing over more than two microscopic fields magnified 500 diameters, but do not ramify to any extent, except at their ultimate portions, where they become knotty and often thickened. Processes from these cells may be found as low as the middle of the barren layer, intermingled with the downward directed processes of the small spindle-shaped cells. There is only a single neuraxôn to these pyramidal cells, which follows a course toward a lateral region and joins some of the smaller bundles of uptending nerve fibres. Collaterals are few in number, and are usually short, coursing toward some of the angular cells of the locality, but seldom can be traced with certainty to them.

A little higher upwards we come upon cells of a form not seen in the lower zones, namely, globular or rounded cells (fig. 26), smooth in outline, but having dendrites with the usual projections, including some longer knobbed ones. The neuraxôn comes off from one of the thicker arms at some distance from the cell body, and waves through the intervening tissues, without giving off collaterals, for considerable distances. Intermixed with these cells are larger ones of pyramidal form (fig. 27). Their bodies are somewhat irregular in contour, the neurodendrites coming off from apex, base and sides, are stout, and soon branch repeatedly, but their length is short, and they are covered with varicose thickenings of some size, ending commonly in a thin pointed termination beyond the last knot. None of the ramifications show the fine arborizations seen in the dendrites of the smaller cells. The apical dendrites may be directed either laterally or upward.

The last type of cell represented in the drawings (fig. 28) is stained only in the highest region of the sections, is pyramidal in shape and closely approximates the size and form of the larger pyramidal cells of the third layer of the cortex. The body is stout, the arms come off from the sides and base by short conical projections gradually attenuating, the apical process is studded with short, thick, lateral projections, and there is just below the free termination a swelling covered with minute hairy extensions. Some of the arms after spreading over considerable distances from the cellular body develop a feathery appearance like similar cells of the cortex. There is only a single axis-cylinder process to these cells arising from one of the thick basal processes and eventually directed upward toward the thalamic regions.

The dendrites of the cells of the infundibular tract, may, as has already been seen, be divided into two classes—neurodendrites that develop knots and varicosities in their course, the extensions themselves being sometimes short, sometimes very extended; and dendrites having short spiculæ and knobbed projections from their sides, occasionally to such an extent as to give them a feathery appearance, and in the latter case they are always long.

The neuraxôns present at their points of origin a diversity of form; they may arise by thin even threads from the cell body or processes, or they may at first be thickened and have a number of large varicosities, or may have a number of minute transverse projections from the thread, or lastly, they may arise by a gradual process of attenuation from a protoplasmic extension, the protoplasm passing insensibly over into the neuraxôn. Some of the axis-cylinder processes of the various cells join the fibres of the nerve bundles by passing into them, and bending into the direction of the run of the fibres at an obtuse angle, or they may, and this is seen with less frequency, join themselves to a fibre of the bundle, the union being made at a T-shaped angle. The very interesting neuraxôns of the intermediary cells have so often been described from other regions of the brain that the figure given in the drawings presents a better idea of their appearance than any further verbal description can possibly add. The network formed by the thin axis-cylinder processes of the cells and the fibres entering the region is very dense, and numerous arborizational endings upon the bodies of the nerve cells are seen in all the sections of the tuber cinerium.

V.—THE NERVE CELLS OF THE CORPORA MAMMILLARIA.

Though not properly included in the infundibular region, a number of sections were made transversely through the bodies of the corpora albicantia, but the forms of the cells only were studied; the other nerve elements have precisely the same aspect as in the regions already examined.

A haze of fine nervous filaments fills all portions of the bodies, encircling the protoplasm of the nerve cells, and following their arms so closely that it is extremely difficult to differentiate the neuraxôns of the cells for any considerable distance from other fine nerve filaments.

Though the boundaries of the corpora are microscopically well defined, nerve filaments, and even the dendrites of some of the cells pass into the surrounding tissues beyond the limits of the bodies in considerable numbers, and between the two bodies a rather extensive system of single nerve fibrillæ pass from one to the other ganglion. The threads of the networks have extensive branchings, and free terminations at the distal extremity of short rami are not infrequent.

There is distinguishable in the inferior half of the bodies a variety of cellular shapes, among them an extremely large, badly stained nerve cell of rounded form, probably corresponding to the unipolar cells lately mentioned by Golgi[1] as located in this region; secondly, well stained triangular and irregularly pyramidal cells of medium size with thick dendrites, the apical one being directed toward the base of the body. The dendrites are of considerable length, but do not branch extensively. Thirdly, cells of rounded pyramidal form with apical processes directed upward and dendrites similar to the last variety. Fourthly, spindle-shaped cells of various forms lying in all directions with dendrites corresponding to the similarly shaped cells of the tuber cinerium. Fifthly, various forms of angular cells with long but not abundantly branched dendrites.

The upper half of the bodies contain spindle cells, but not in the same proportion as in the lower region, small pyramidal and irregular cells, and a large pyramidal cell, the largest stained nerve body in the regions studied. This cell is of irregular shape, the protoplasmic processes thick and short, with numbers of thinner dendrites coming off from them, giving a bushy effect to the entire cell. The axis-cylinder processes of these cells are at first directed downward, then laterally and inwardly, and appear to conjoin with the fibres of some of the numerous bundles located along the inner margin of the bodies.

---

[1] *Berliner Klin. Wochensch.*, No. 14, 1894.

The dendrites of the majority of the cells of the corpora mammillaria are moderately long, knotty, and have the short projections from their sides, but none of the feathery arrangement of the cortical cells and terminate in an attenuated free ending. Occasionally the termination is more complex, and similar to certain of the spindle-shaped cells of the tuber cinerium.

Though the nerve cells of the corpora are fully tinged, no neuroglia cells can be seen stained among them, though in the tissues adjacent to their edges, and along the margin of the tuber the same sustentacular cells of the more anterior region are found, and above them are numerous mossy and long-rayed Golgi cells.

EXPLANATION OF THE DRAWINGS.

FROM THE PITUITARY GLAND.

FIG. 1.—A portion of a follicle from the anterior lobe of the pituitary, with a nerve fibre crossing from the adjacent territory over the vessels surrounding the follicle, and finally branching extensively upon its epithelial cells. The terminations of the branches of the nerve fibre are all in knob-form, and apparently are situated between the epithelial cells.

FIG. 2.—Vertical section of the posterior lobe of the pituitary, the various elements arranged somewhat diagrammatically; *a*, *a*, capsule of the lobe thickened in places, from which extend threads that end in knobs at an inner line of separation from the more centrally lying cellular elements; *b*, *b*, rows of epithelial cells arranged after the manner of the cuticle; *c*, inner limiting line; *d*, *d*, transversely cut follicles of the posterior third of the lobe, lined with cubical epithelium; *e*, connective tissue trabeculæ; *f*, epithelial cells of irregular formation; *g*, lowermost group of ganglion cells; *h*, *h*, unstained groups of nerve cells; *i*, *i*, terminal dendritic tufts; *k*, medio-anterior nerve cells of irregular pyramidal form; *l*, *l*, neuroglia cells of the anterior border; *m*, tufted cells, *n*, cells belonging to the fifth group of the text, *o*, nerve cells

of different forms along the superior border; *p*, *p*, feltwork of fine varicose nerve fibrillæ along the superior border of the lobe; *r*, nerve haze of the central and anterior regions; *s*, free arborizations of fibres coming from the central regions, situated between scattered nerve and other epithelial elements of the region; *t*, rounded thickening in the course of a nerve fibre, near which is also a flask-shaped projection from a fibre; *v*, *v*, peculiar tufted endings of nerve fibres upon rounded and oval bodies of the central region of the lobe; *w*, smaller pyramidal nerve cells. The lobe is enlarged 34 diameters, the various elements 90 diameters.

FIG. 3.—A mossy neuroglia cell with large body from the anterior portion of the posterior lobe of the pituitary.

FIG. 4.—A long-rayed neuroglia cell from the same locality.

FIGS. 5 and 6.—Cells of the first sub-class of irregularly pyramidal and oval forms, having large numbers of dendrites terminating in feathery arborizations, and a single neuraxôn.

FIGS. 7 and 8.—Cells of the second sub-class with a single long protoplasmic extension directed toward the superior border of the lobe.

FIG. 8—Flask-shaped cell of the third sub-class with smooth dendrites and several neuraxôns.

FIGS. 10 and 11.—Small cells of the fourth sub-class, with exceedingly long pyramidal extensions directed upward.

FIG. 12.—Cell of the fifth sub-class, with numerous short hairy and feathery projections from the dendrites.

FIG. 13.—Very small tufted cells of the sixth sub-class, with a bush of smooth wavy apical dendrites.

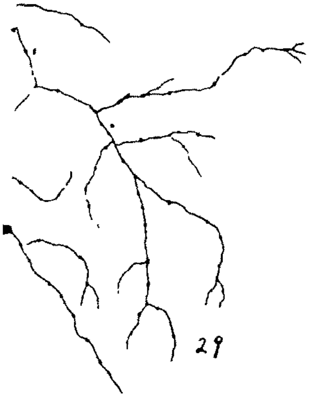
FIG. 14—An intricate termination of two dendrites of a cell of group *g*, at the base of the lobe, highly magnified.

FIG. 15.—A portion of one of the peculiar terminations of the neuraxôns upon one of the oval bodies situated in the central regions of the posterior lobe, highly magnified.

FIG. 16.—One of the oval bodies from a similar situation surrounded by nerve fibres, which give off lateral branches, ending in peculiar tufts and comb-like figures.

FROM THE INFUNDIBULUM.

FIG. 17.—Semi-diagrammatic drawing of a transverse vertical section of the infundibular region of the brain, at the opening of the cavity of the ventricle into that of the infundibulum; *a*, lumen of ventricle; *a'*, lumen of infundibulum. 1, Primary forms of ependymal neuroglia, the processes extending from a cell-like body at the edge of the ventricular cavity to the sub-pial limit. 2, Coarser and less ramified variety of ependymal cell. 3, Coarse ependymal cells, branching within the inner half of the infundibular wall. L, Portions of ependymal cells with tufted sub-pial branchings. 5, Unstained nerve cells. 6, Pyramidal cells with long fine processes. 8, Transversely lying cells of small size with knobbed extremities. 9, Pyramidal cells with large numbers of apical processes. 10, Probable axis-cylinder extension of pyramidal cells with thickenings, and rectangular extensions to the sub-pial limit. 11, Nerve fibres passing from the infundibular wall into the tissues along the border of the ventricle. 12, Burr-like cells of the infundibular wall. 13, Line of the floor of the brain. 14, Long-rayed ependymal



29

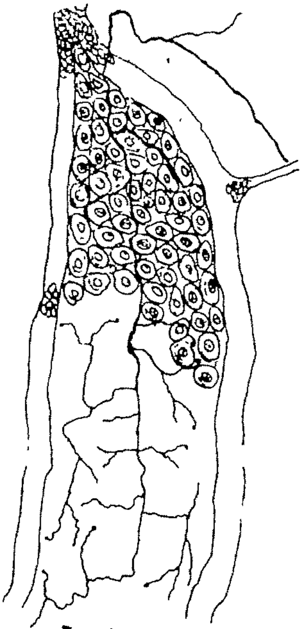
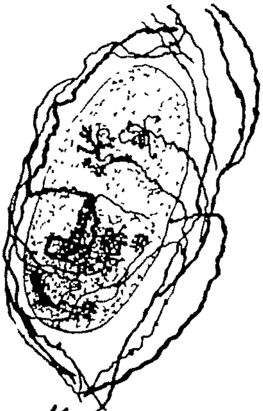
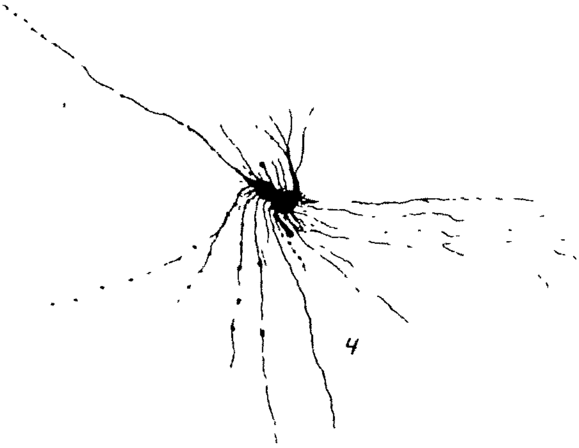


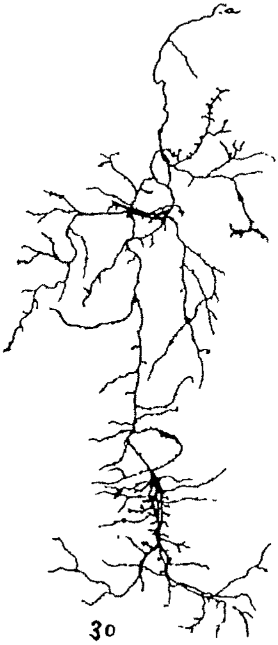
Fig 1



16



4



30

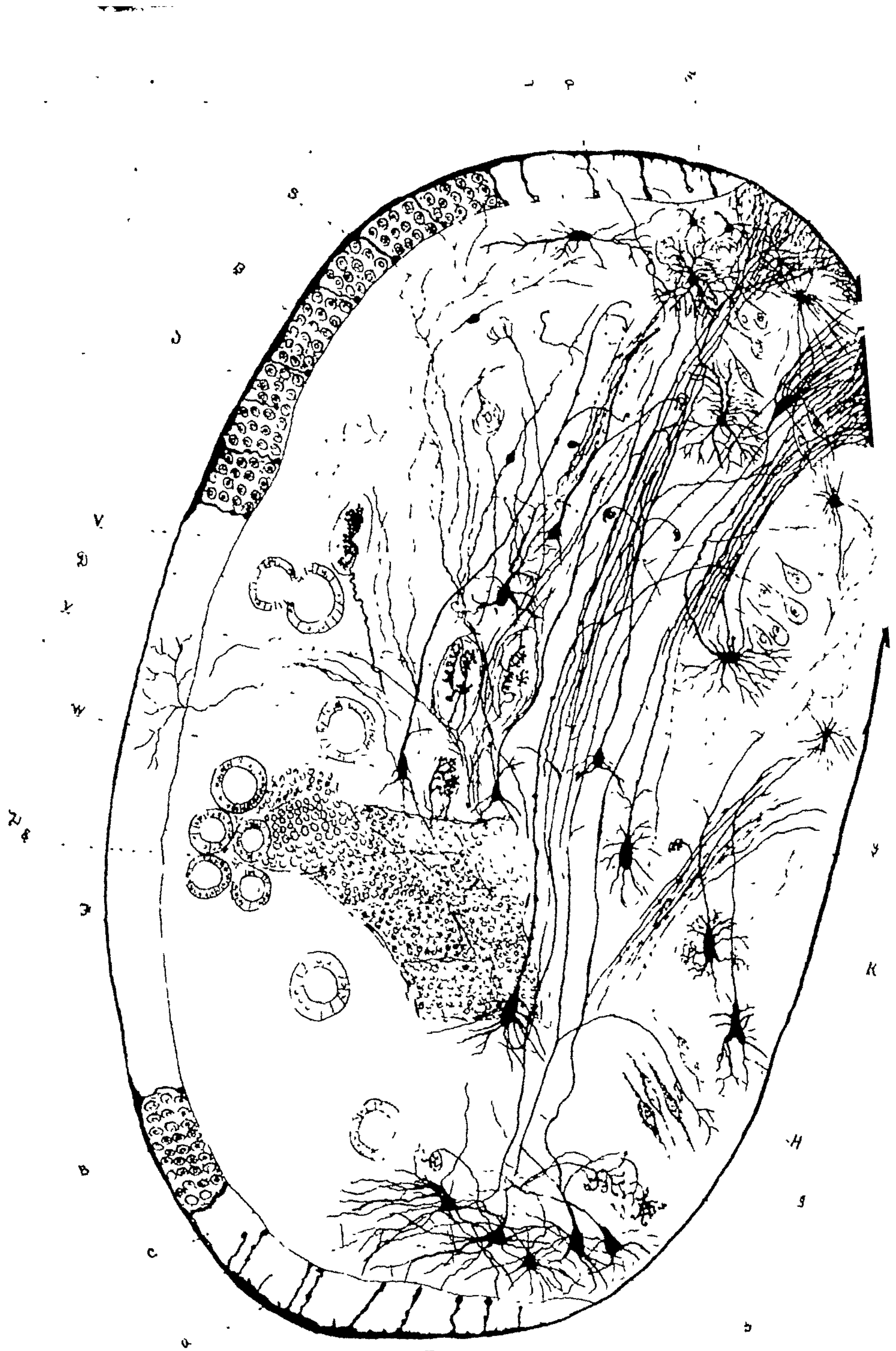
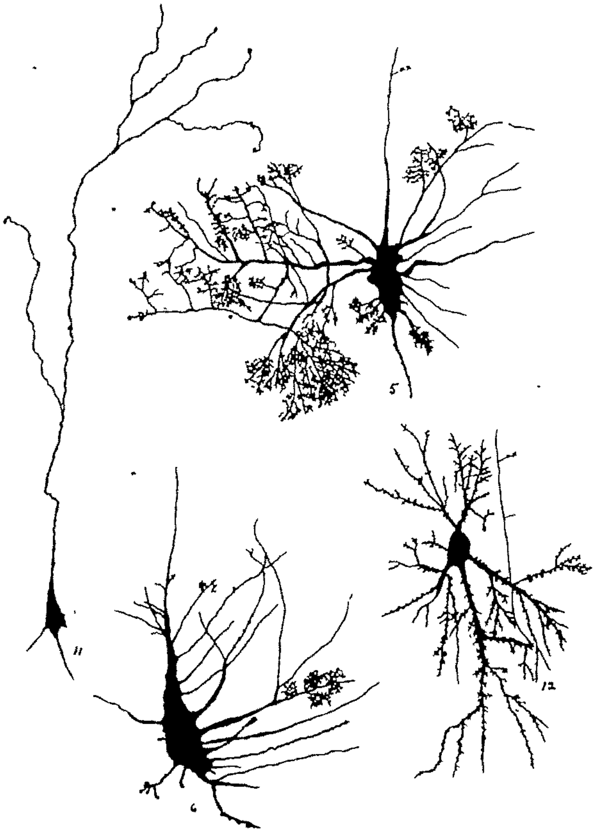
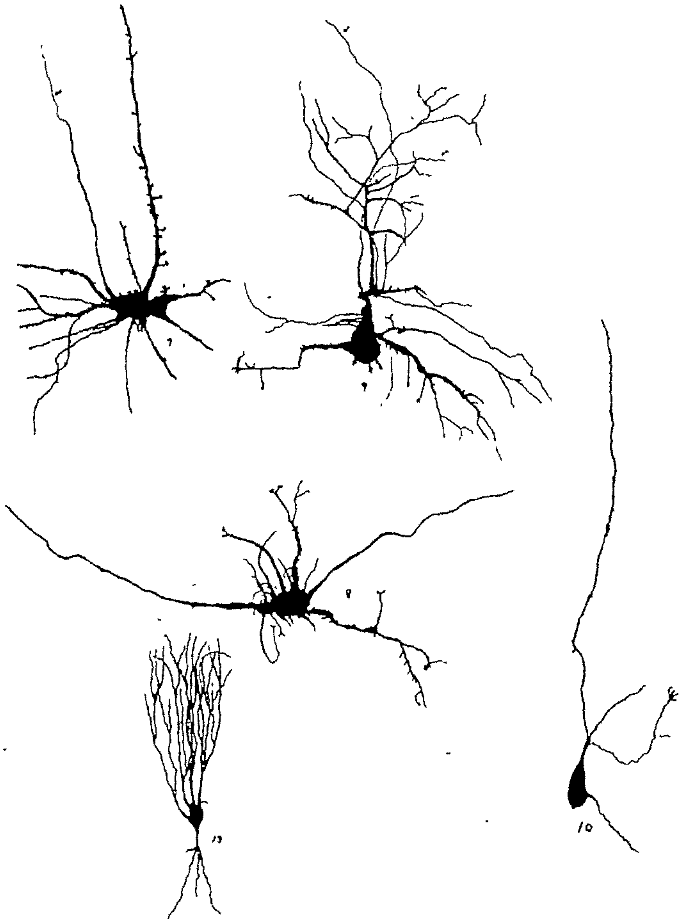
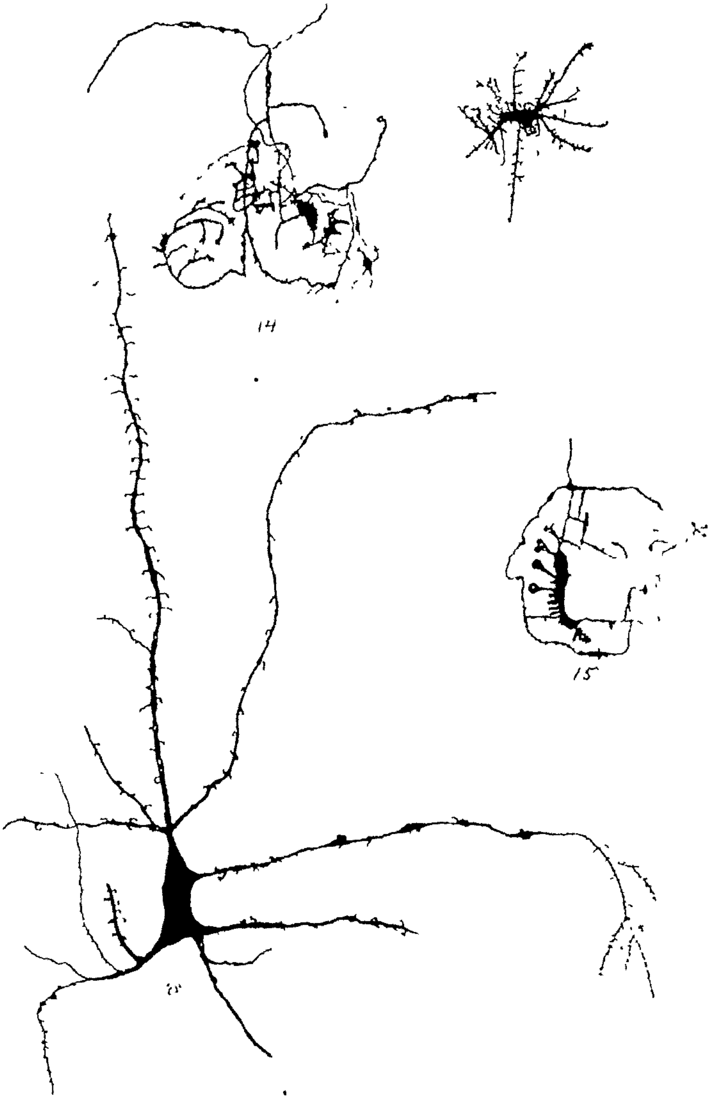
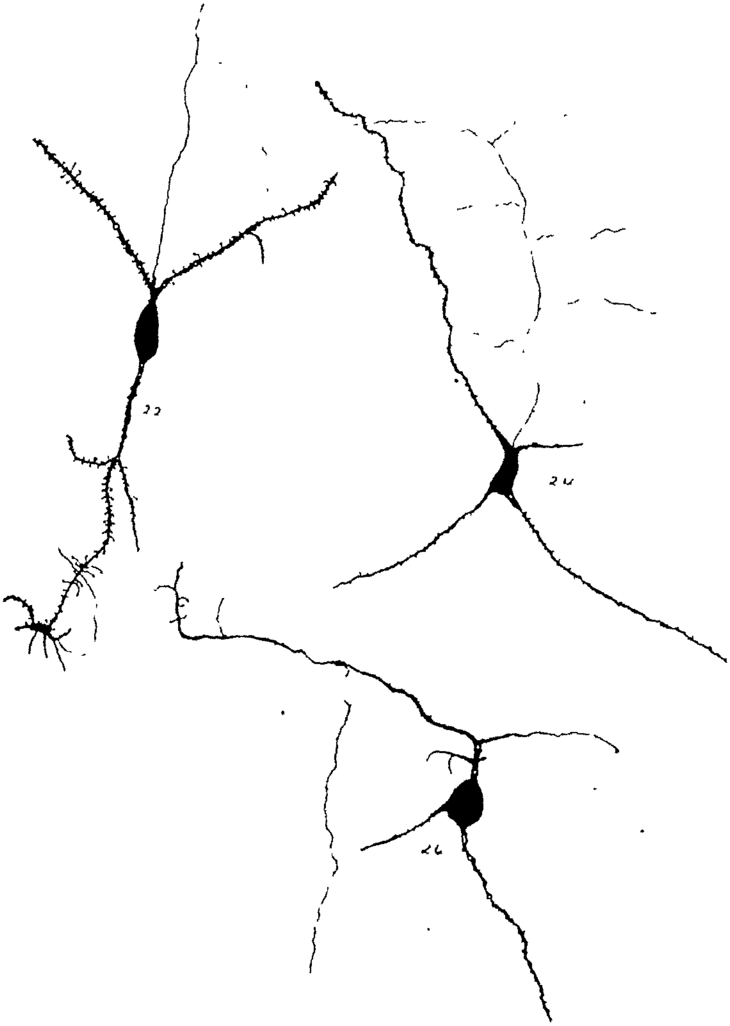


Fig 2.


5
11
6
12


7
9
8
10
13


14
15
13


22
24
26

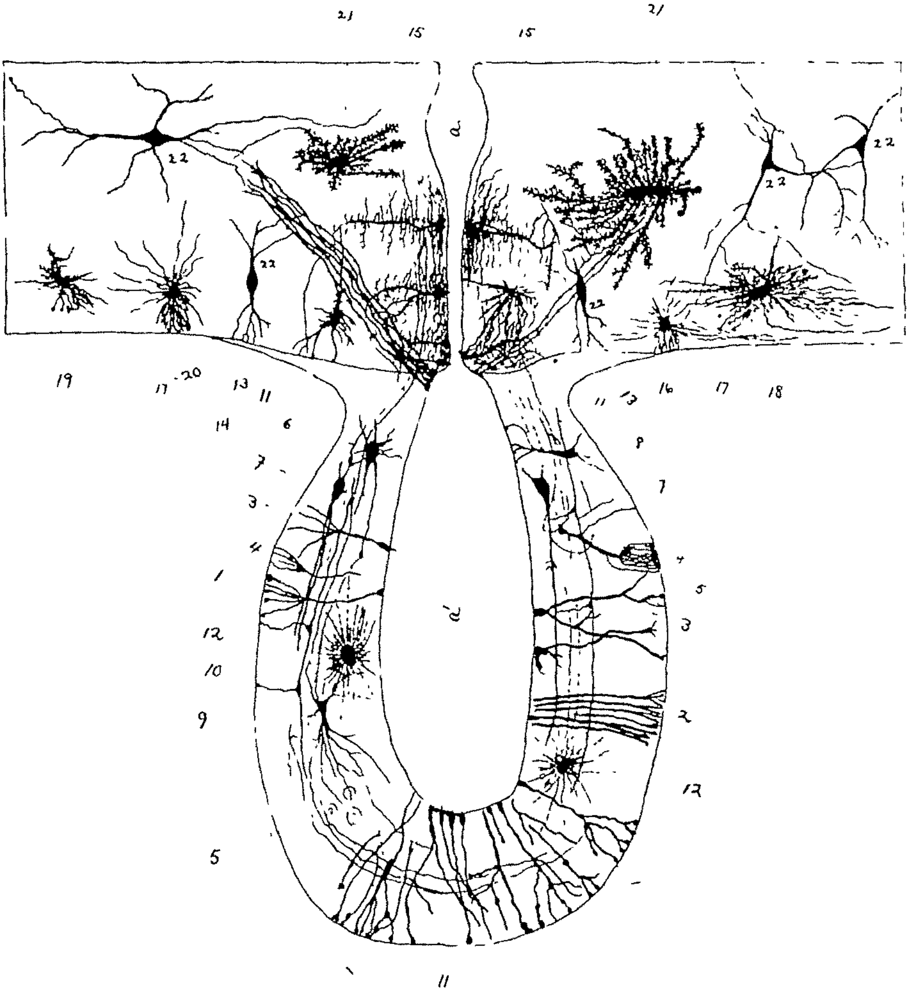
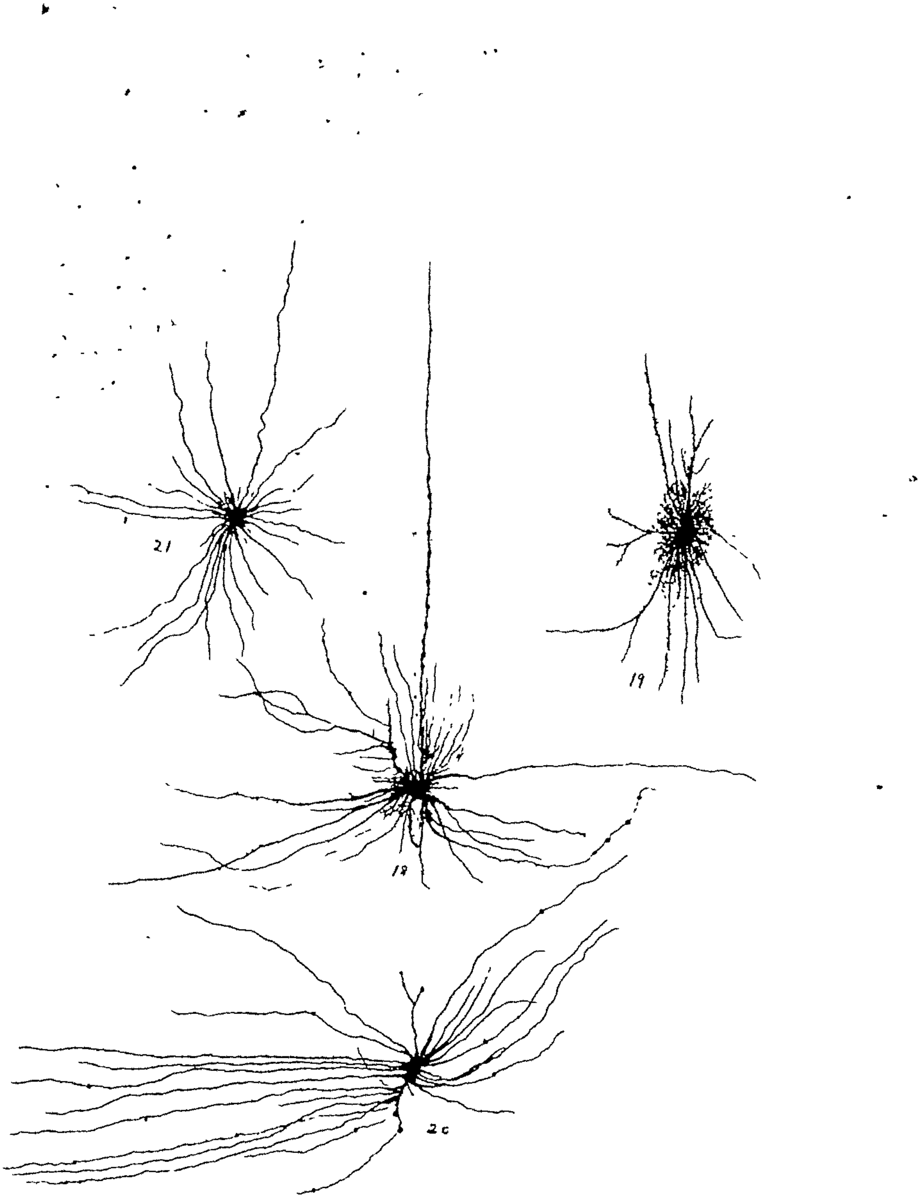
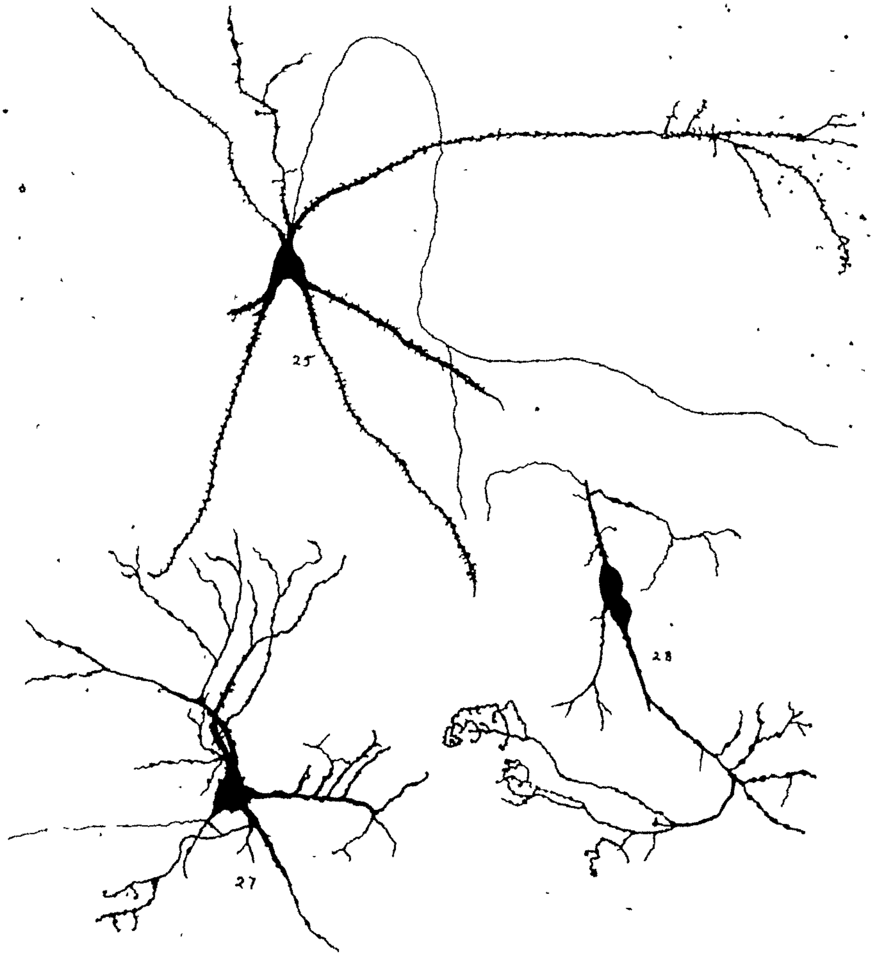

a
17 · 20
a'

Fig. 17


21
19
18
20


25
27
28

cells of the juncture of the ventricular and infundibular cavities. 15, 15, Fir-tree ependymal cells of various sizes and forms lining the border of the ventricle. A few of them are seen to have rounded knobs adjusted against the pial limit of the basis cerebri. 16, Neuroglia cell approximating the short-rayed type of Golgi. 17, 17, Sustentacular glia cells of the inferior border of the tuber cinerium. 18, 19, Glia cells with numerous long and stout hairy processes from the bodies and thicker projections, probably transition forms between the sustentacular cells, and cells of later development. 20, Probable nerve cell resembling some of the glia cells. 21, 21, Large mossy cells situated at some distance from the ventricular border. 22, 22, Nerve cells of different forms.

Fig. 18.—A large sustentacular glia cell from the base of the brain, with long, knotty, upward extension.

Fig. 19.—A hairy cell from the floor of the tuber.

Fig. 20.—A stout neuroglia cell with extremely long-beaded rays from the border of the optic nerves.

Fig. 21.—A long-rayed Golgi cell from over-lying regions, drawn for comparison to show the extent of the tentacles.

Fig 22.—Spindle-shaped nerve cell with smooth body and thick arms. The lower dendrite ends with a peculiar figure having an axis-cylinder process arising from one of the limbs.

Fig. 23 —Spindle-shaped cell of another type having very much beaded dendrites.

Fig. 24.—Angular cell with uptending branched axis-cylinder process.

Fig. 25.—Irregular pyramidal cell with long branched apical process, and single axis-cylinder, coursing laterally.

Fig. 26.—Rounded cells with smooth outlines.

Fig. 27.—Pyramidal cell with short knotty processes, and a single axis-cylinder.

Fig. 28.—Pyramidal cell approximating in shape cells of the third layer of the cortex.

Fig. 29.—Outspread termination of one of the collaterals of a spindle-shaped cell, near the inferior layer of the tuber.

Fig. 30.—A more complex termination of one of the angular cells of the region. At A, a single nerve thread is seen, which breaks up into intricate figures.

ON SOME OF THE NEWER ASPECTS OF THE PATHOLOGY OF INSANITY.

BY W. LLOYD ANDRIEZEN, M.D.LOND.
Pathologist and Assistant Medical Officer, West Riding Asylum.

INTRODUCTORY.

THE study of the central nervous system, its origin, and the evolution of its structure and functions, and their correlation to the actual life of the organism is interesting to every student of Nature, and not merely to the neurologist or the alienist. Between the latter two classes of workers a gap has hitherto existed: the neurologist pure and simple, mainly restricting himself to the study of "nervous diseases" and their elucidation, based on general researches in neurology and having no distinct relation apparently to insanity, has lived and worked in a world by himself: the alienist in a more isolated, and also more easy-going fashion, having mainly to deal with so-called "mental disease," the phenomena of which he naturally supposed could be unravelled by metaphysical and psychological speculation, went further out of touch with the neurologist, and contributed to further widen the gap between these two.

The recent growth of a more scientific spirit of inquiry in psychology after the methods and spirit of Darwin, and the more recent and important neurological discoveries since 1870, have slowly tended to bring more and more together these two branches of study. The gradual recognition of the inadequacy in the methods of the older metaphysicopsychological writers, and the increased interest in the study of the brain and nervous organs themselves by various physiological and pathological methods, and the further feeling that an attempt should be made to correlate these with the actual activities of life, growth, and conduct of the individual —all these are slowly working towards the desired result.

Under the old *régime*, which is not yet dead, insanity cannot be said to have fared well or worthily. For even so recently as half a century ago it had been looked upon with aversion and horror, as a thing apart in the domain of medicine, a thing horrible and analogous to demoniacal possession (as, *e.g.*, epilepsy, which was called morbus sacer, the holy disease, by the ancients), and its early treatment by exorcism, holy water, stripes and chains, immersion, and other methods, were the natural outcome of ignorance and superstition till Philippe Pinal in France, and after him William Tuke in England, inaugurated the beginnings of a more gentle and humane system in the treatment of the insane.

But little progress could be said to have been made in the study of insanity and its treatment, till physicians came to look at it in precisely the same way as they did ordinary disease; to study mind as a brain function which is found in nearly all animals in varying degrees; which in man arises from small beginnings like any other function, then gradually develops and attains the acme of its complexity and activity in adult life, and finally fails and disappears with the decay of old age—in a word, as distinctly correlated with the anatomico-physiological development, growth, and decay of the brain and nervous system.

Later discoveries in physiology pointed more strongly to the same conclusion, especially since the representations of certain bodily functions (higher sensations and perceptions, muscular movements, and complex acts of conduct) came to be located in the brain, and their intimate association with one another, and with mental activity shown to exist. Biological studies in the development and life histories of nervous systems throughout the animal kingdom, the sociological study of heredity as influencing mental like other disease, have all contributed to show that the insanities, like other variations in the organism, are subject to natural selection, and may be perpetuated in the race through favouring causes; that in this way hereditary qualities and potentialities for evil or good leave their seams and tracts in the organisation of the human brain, rendering it potent under suitable stresses of the environment to issue in insanity, vice, and crime. This aspect of the problem I have discussed elsewhere.[1]

The most reasonable hope, therefore, of obtaining knowledge seemed to lie in our investigation of the insanities, not merely as so many complexes of psychical symptoms—the short and easy method of the symptomatologist and speculator—but in their relation to the known and growing facts of neurology, both neuro-physiology, neuro-histology, and embryology, as applied to the further study of the brain, including its evolution and dissolution in man and animals. Such work has only been possible since the last quarter of a century.

Bases for Study.

Four great factors have contributed to the building up of such a foundation on which alone the scientific study of the insanities can be really based, and these may be passed in review in chronological order.

The first and earliest, the doctrine of the localisation of function in the cerebral cortex of animals and man, beginning with the researches of *Broca* before 1870, and of *Fritsch* and *Hitzig* in 1870, has opened up a new world in the obscure region of brain function; and its further development and cultivation at the hands of Ferrier, Munk, Horsley, and Schäfer, has yielded results of the utmost promise in the successful diagnosis and treatment of many gross diseases of the brain with which must ever be associated the names of Hughlings Jackson, Horsley, MacEwen, and others too numerous to mention.

---

[1] "On Insanity and Race Decay" (discussion opened at the annual meeting of the Medico-Psychological Association of Great Britain and Ireland, in Dublin, June 13, 1894).

The second, the doctrine of the three evolutionary levels of the central nervous system (including the evolutionary sub-grades of the cortex), and the attempt to apply the doctrines of evolution and dissolution of functions, according to what would seem to be a certain natural law to these parts of the nervous system, has furnished such a valuable key to the clinical study of so many brain diseases generally, that its vast importance cannot be over-rated ; a doctrine which we owe to the laborious investigations and profound insight of Hughlings Jackson : one which furnishes us with a valuable weapon in investigating the onset and course of march of some of the insanities—notably the epileptic, alcoholic, and the progressive paralytic—a key greatly utilised in the two recent works of Lewis ("Mental Diseases," 1890) and Mercier ("Sanity and Insanity," 1891), on insanity. The results of this are, however, as yet in their infancy.

The third and in many ways the most wonderful, inasmuch as it helps to come nearer to the psychical by the pathway of the neurological and to penetrate and understand the most intimate mechanisms and workings of nerve centres, must be associated with the doctrine of the "neuron," the unit of nervous and psychical function considered from every point, whether anatomical, physiological, embryological, psychological, or pathological, a doctrine developed on the anatomico-physiological and embryological side by the immortal work of Golgi and of Ramon y Cajal, and on which some confirmatory experimental work already done by Horsley and Gotch calls for notice ; a discovery which is vast and epoch-making in character, already revolutionising our old ideas of the mechanism and mode of action of nerve centres, and which must influence all future work in neurology, psychology, pathology, and the insanities generally.[1]

The fourth and last, the comparative and sociological method, has again as yet received scant recognition, though here again important light is certain to be thrown in the near future in the investigations of *idiocy, imbecility,* cretinism, and ethnic and atavistic types of degenerations, including the study of the criminal and of criminaloid, vicious and paranoiac types. In the present communication I shall deal mainly with the third great branch of study, especially in relation to the newer light which I think it throws on the pathology of insanity.

---

[1] The first recognition of it in an English work should be here recorded, *i.e.*, the Morison lectures by Dr. Batty Tuke on "The Insanity of over-exertion of the Brain," 1894.

Old Views.

The structure of the brain and its cortex, and similarly of the other nerve structures, has hitherto been held to consist of a network of nerve cells and nerve fibres, with processes partly anastomosing (Schroeder Van der Kolk, Gerlach), and partly merging into and becoming fused in a granular mass, the basis substance—a region therefore in which, according to the language of *Rindfleisch* (*Centralblatt für die medicinischen Wisenschaften,* 1872, p. 77), the "fibrillary" and the "granular" structures are fused. Thus the connections between neighbouring nerve cells was effected in two ways: (*a*) by direct anastomosis of certain processes (see fig. 1 from Schroeder Van der Kolk) and (*b*) by the merging and fusion of certain other processes into a finely granular or amorphous mass, *i.e.*, by the methods respectively of *anastomosis* and *fusion* with the "granular substance"—an intercellular substance, therefore, common to all the various nerve cells and of an intermediate nature.

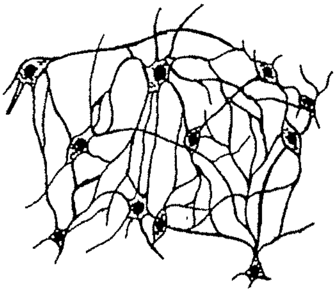


FIG. 1.

Shows Van Der Kolk's plexus of nerve cells, anastomosing by their processes one with another.

Such were the two dominant conceptions which prevailed with regard to the constitution of nerve centres. The intra-central course of the sensory nerves in the spinal cord, and their mode of ending had been unknown, but speculations were not uncommon respecting their *supposed* endings, and their relationships to the cells in the spinal cord. The same ignorance on a point of such fundamental importance may be said to have prevailed—indeed, more so—with regard to the higher centres in the brain cortex. The modes of investigation then in vogue, viz., with the carmines (Gerlach), picro-carmine (Ranvier), hæmatoxylin, the anilines, osmic acid (Exner), and the gold-stains (Gerlach, Freund) failed to unravel the mystery, and it seemed as if the question would never be solved. So it remained till Golgi with his precious method entered the field, and gave us a means whereby the question could be definitely settled once for all, and other light also obtained on the structure and constitution of nerve elements which till then it seemed vain to expect.

In the present inquiry it will perhaps be more profitable to begin with a consideration of the simpler kinds of nerve centre before leading up to the infinitely more complex organisation of the human brain cortex itself.

Comparative Neurology.

In (*a*) *the lowest type of nerve centre* such as we first find realised amongst the coelentera (hydra, certain medusæ, &c.), the nerve apparatus is exceedingly simple. In the lower class of the unicellar protozoa all microscopic research has so far failed to demonstrate the presence of nervous structures. In them one and the same cell-body is able to exhibit the various phenomena of response to stimulus by contractile movements much as a leucocyte or other unicellular organism could. The power of response to external stimulus is the property of all living protoplasms whether animal or vegetable. But when we proceed to the next grade of the animal kingdom, the *Metazoa* (multicellular organisms) we find a specialised system of cells set apart for the reception of and response to external stimuli. In these animals we have for the first time a physiological division of labour among the constituent cells, some being *protective and sensitive* (ectodermal), others *digestive* (endodermal) and others *reproductive* in function, &c. Corresponding to this physiological differentiation of function there is a morphological differentiation of structure which can easily be recognised by the microscope; and amongst such morphologically differentiated tissues we find the first evidence of distinct nerve-elements in the animal kingdom as shown by *Romanes* (" Star-fish, Jelly-fish and Sea Urchins:" a research on primitive nervous systems, 1880); by the brothers Hertwig (" On the Sense Organs and Nervous systems of the Medusæ," 1880); by Exner, Schäfer and other workers during the period from 1882-90.

Among these Metazoa we shall study both hydra and the medusæ. *The nerve mechanism* in hydra is very simple. In and among the cells of the ectodermal layer are certain specialised cells the free border of which is furnished with tactile (sensitive) hairs, with their deeper parts attenuated and prolonged into thread-like processes which pass into the thin sheet of muscular or contractile cells which lie between the ectoderm and the endoderm (fig. 2 B). We have, therefore, the provision here of a sense-organ, *i.e.*, a sensitive nerve cell in its elementary state, being none other than a specialised epithelial cell, the superficial portion of which is the recipient of sensory stimuli from without, and which by its deeper part or process communicates the excitation (in the direction of the arrows in fig. 2 B) to the subjacent contractile tissue, with the result that a movement follows the stimulus. In the *medusæ* the nerve cell (sensitive cell) is placed also in the ectoderm, and the various nerve cells have a further tendency to arrangement in a *ring* of nerve cells and fibres along the margin of the swimming-bell. These send a deep fibre each to the subepithelial tissue in which other *nerve cells* (ganglionic plexus) are imbedded. Whether these fibres (➳ fig. 2 C) enter into continuity with one of the ganglion cells of the plexus (*c.b.*, fig. 2 C), or merely comes into *contact* with such a deep-lying cell is not certain, but there is no doubt that the nervous excitation affects this deeply placed ganglion cell of the plexus. From this latter cell issue one or two processes (➤→ fig. 2 C) which end by coming into contact with the muscular cells which form a sheet immediately underlying the ganglionic plexus. The progress of the nerve current is thus along (*a*) *epithelial sensitive* cell and along (*b*) its *basal process* (nerve-fibre), to (*c*) the *ganglion cell* of the nerve plexus. From the latter the current issues along its processes (*d*) *or fibres* to the contractile tissue or muscle fibre. We shall later on have occasion to point out that the *peripheral sensitive* cell is bipolar, and is found in all animals from the medusæ upwards to man; and in the human fœtus it is developed not from the central medullary plate but from the

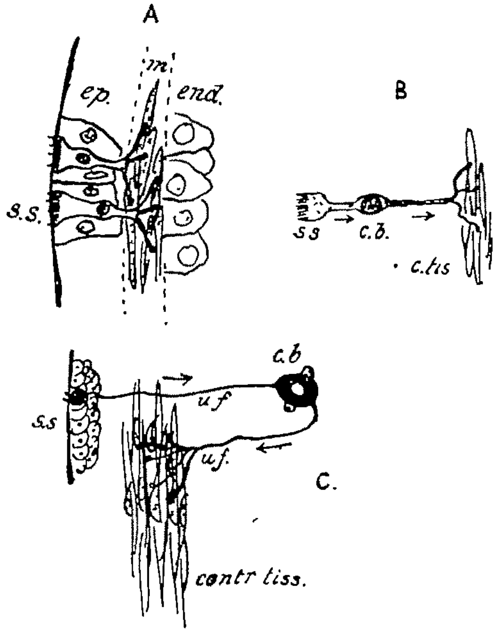


FIG. 2.

Lowest reflex types of neurons (hydra and medusa).

A. Body of hydra. *ep* Layer of epithelial cells, the surface of which *ss.* is sensitive. The basal part of two cells shows division into three processes each, distributed amongst *m*, the middle layer of contractile tissue. *end.* Endoderm (digestive cells).

B. Neuron (neuro-epithelial cell) of hydra shown diagramatically.

C. Neuron of *medusa* (a more highly developed animal).

lateral ridge (Zwischenstrange of His); and that while its distal prolongation ends in the superficial cutaneous epithelium, its central prolongation passes into the spinal cord.[1] In fact, it may be said to be the first and most fundamental structure in the development of a nervous system, and in the simple body of an organism like *hydra* is the only neuron present.

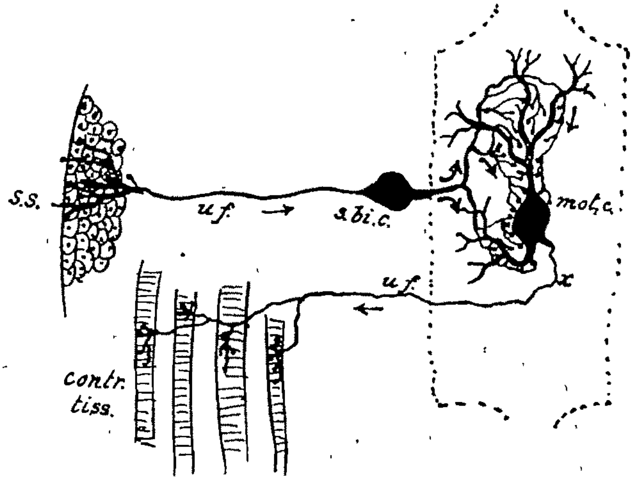


FIG. 3.

Higher Reflex Type of Neurons as in higher invertebrates and lowest vertebrates.

s.s. Sensitive surface, in which the nerve-fibre ends.
u.f. ➤→ afferent nerve-fibre, going to
s.bi.c. Sensitive bipolar cell.
↳→ Distribution of the nerve current in the direction of the arrows
↱→ within the nerve centre.
mot. c. Motor cell from which issues at x another
←◀ *u.f.* Nerve fibre (efferent), which goes to
contr. tiss. Contractile tissue.

But as we ascend a little higher, *i.e.*, above the level of hydra, we get a differentiation of the neurons into two distinct types: (*a*) the peripheral sensory neuron; and (*b*) a *motor neuron* connected with its appropriate muscle (fig. 3). These two constitute the fundamental type of nerve centre for all the vertebrates from amphioxus up to man.

---

[1] In a future publication of the author's these homologies and other points of importance will be fully described and illustrated.

The noteworthy feature is that the *bulbo-spinal axis* of all vertebrates is composed of such an aggregate of neurons forming really a *bilateral chain,* the two halves of which, however, in vertebrates meet and become connected with one another in the mid-dorsal line of the body forming the neural tube.

In what way do the *sensory and motor neurons* come into relation with one another within the bulbo-spinal axis? Is there a direct anastomosis of the processes of the one with those of the other—like an arterial stem opening out and branching into capillaries, which then re-combine into a single venous tube? Or is there an intermediate substance into which the processes merge, gradually losing their individuality and *blending* with the common substance? Or do processes of the one simply come into contact only with those of the other, sufficiently thereby to transmit the nerve currents? Anatomical investigation with the most refined methods at present available, notably the method of Golgi, enable us to give a definite answer to the question, and to state that the last named is the method whereby the passage of the nerve-excitations is effected from the one neuron to the other; viz., by *contact.* Let us add that, however fine and delicate the ultimate divisions and subdivisions of the processes of the nerve cells may be, they ultimately end freely: there is no merging into or blending with any intermediate substance; but each branch maintains its individuality to its ultimate parts—to its very finger tips, as it were. The anatomical and physiological integrity and individuality of the neuron is thus a fact, and one of fundamental importance, one which should be ever kept in mind, for as we trace the complexity of nerve structure up to the level of the highest of all nerve structures—the cerebral cortex—we shall find this fundamental feature preserved throughout. Let us further add that we owe the importance of this teaching to the embryological work of *His*, of *Forel*, of *Kölliker*, and of *Ramon y Cajal*, the last of whom has furnished the most conclusive and indisputable proofs in support of the doctrine in question. The above description, together with the illustration in fig. 3, do not exhaust the facts with regard to the nervous mechanisms in the bulbo-spinal cord. For as we know from biological investigation, the vertebrate spinal cord is composed of a series of ganglia in two chains (bilateral) which, however, early in the life history of vertebrates, fuse together in the middle line. According, therefore, to the serial or linear connections of the several ganglia one with another, we would naturally look for *connective neurons* or fibres within the spinal cord as well (inter-ganglionic or association fibres), while underneath the tranverse connections we should expect to find longitudinal *neurons* or fibres also. In other words, we would be prepared to find, not only the fundamental (sensory and motor) neurons of the bulbo-spinal system, but an accessory system of inter-connecting intrinsic neurons, as well for the co-ordination and sequence of reflex movements which the spinal cord is up to a certain extent capable of effecting. Having stated thus much by way of preface, let us now pass, therefore, to the minute investigation of the bulbo-spinal system in the *higher* vertebrates (*i.e.*, above the fishes). In the spinal cord of the kitten, which I have chosen for investigation because it is an animal sufficiently high in the mammalian scale to enable us to deduce conclusions applicable to man, we may begin with the entry of the posterior root into the spinal cord. My investigations have been conducted on kittens of the ages respectively of one, three, five, ten, fifteen, twenty-one, and thirty days after birth, all stained and prepared after Golgi's method, and sections cut transversely and longitudinally. At the outset let me acknowledge my especial indebtedness to the work especially of Ramon y Cajal in this connection, and the admirable way in which he has cleared the ground for subsequent workers. Much of what I shall have to say will, I am afraid, be but of secondary interest, though perhaps of some little value as illustrating the ground plan and some of the superstructures which run through the mammalian cord. Let me state at the outset that by the term bulbo-spinal cord or axis is meant the whole system of metameric centres starting in front from the nuclei of the oculo-motor nerve (third cerebral nerve), and ending behind with that of the last sacral nerve. Such a bulbo-spinal axis represents the fundament of the nervous system, and is to be looked upon as a tubular structure (open at both ends) which in ancestral vertebrata, as also in certain existing acraniates, had two communications with the exterior in front and behind : in front by an orifice, the dorsal neuropore,[1] and behind by a tubular canal, the neurenteric canal. Both these apertures, however, quickly close, and in craniate vertebrates they can only be observed as a transient phase in early embryonic life. The side walls of this tube contain among its epithelial cells some which are destined to increase and multiply rapidly (neuroblasts), and to give rise to the nerve cells ; while others develop a system of supporting fibres which form a scaffolding (spongioblasts) surrounding and supporting the growing nerve cells. This is the central or axis system. But another factor soon makes its appearance. Certain epithelial bipolar-cells from an epiblastic ridge adjoining the lips of the neural tube (Zwischenstrange) now send in processes (nerve fibres) which *invade* the central tube, and come into intimate contact with the processes and cell-bodies of the intrinsic cells of the neural tube. This bipolar cell, one of whose processes grows into and enters the neural tube, while the other ends on the surface of the animal's body, is the cell of the posterior spinal ganglion in man—bipolar in the human embryo up to about 2½ months, but becoming unipolar later on. The bipolar condition also occurs as a transient phase in all vertebrates above fishes (*e.g.*, embryo-chick, reptiles, mammals); in the fishes themselves the bipolar condition is permanent and persists into adult life. In a classification, therefore, of the neurons in the general nervous system, we have a fundamental distinction into (*a*) the *primary* or *peripheral sensitive neuron* (epithelial in origin, developed from the Zwischenstrange topographically, bipolar in character, and with a deep process entering the bulbo-spinal axis—posterior root); and (*b*) the *neurons within the bulbo-spinal* axis (primary central neurons). The latter fall into two great groups, *e.g.*, first, *motor or indirectly motor*, and second, *other* types. The *first class* is distinct and well-defined. The *motor* neurons are those which are directly connected by a process or fibre (motor-fibre) with the *contractile* tissue (*c.f.* figs. 2, 3 and 4); they exist in all the bulbo-spinal nuclei from the oculo-motor

---

[1] An account of its significance and life history is given in my article on "The Morphology, Structure, and Evolution of function of the Pituitary Body, and its special relation to the Central Nervous System," *British Medical Journal*, January, 1894.

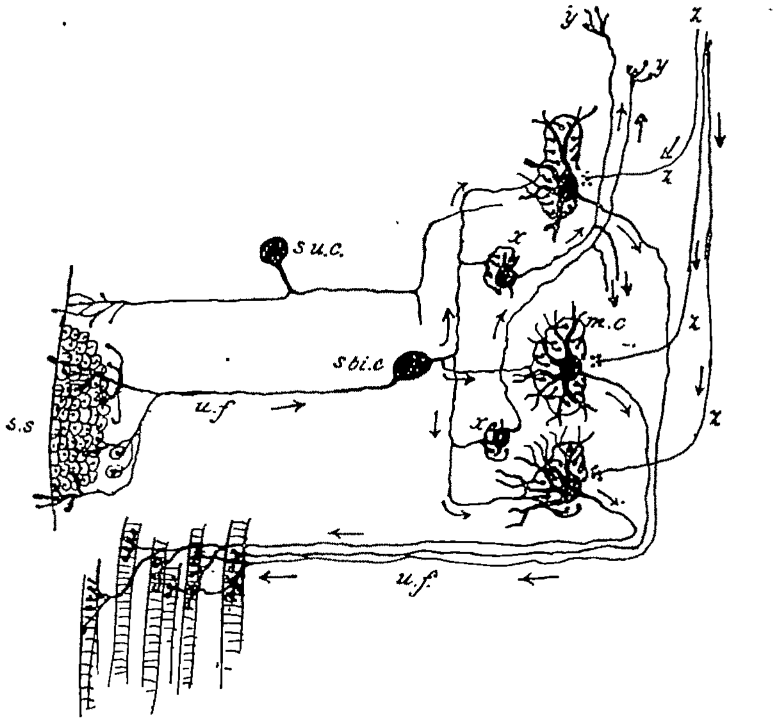


FIG. 4.

Highest reflex type of neurons.

(Spinal cord of fishes, amphibians, reptiles, birds, and mammals.)

s.s. Sensitive surface.
u.f. ⊳→ Afferent nerve (sensory).
s.bi.c. Sensitive bipolar cell.
m.c. Motor cell.
←⋖ *u.f.* Efferent nerve (motor, &c.).
x. Other nerve cells with nerve fibres going to *y*, *i.e.*, other parts of the nervous system. (These cells may be *associative*, *commissural*, or *projection*).
z. Nerve fibres coming from other parts.

region to the lower end of the cord. Whether they innervate the motor organs of the limbs, trunk, viscera, or blood vessels are matters of detail : the essential feature is that they pass into *contractile* tissue, and that by discharging their current into the latter, cause *muscular* contraction. We have used the phrase motor or indirectly motor : the latter part requires explanation. The act of secretion from a gland, or the discharge from an electric organ can hardly be called motor in the conventional sense. Yet we may call these "indirectly motor." In the Torpedo, a tissue which is analogous to modified muscular elements, and in Malapterurus, a tissue which is modified glandular tissue, is innervated from cells which are the exact homologues of true motor cells. The difference lies in the fact that the peripheral *re-acting body tissue* (modified muscular in the one case, and modified glandular in the second), instead of re-acting by muscular contraction or a discharge of secretion, responds by an electric discharge in these fishes. Such electric discharge, however, may be justifiably included in the category of "motor" effects, or "indirectly motor." Leaving, however, the group of indirectly motor neurons out of our present consideration, the motor neurons form a well defined homogeneous group, arranged in serial and segmental order in the bulbo-spinal tube. The primary correlation of these with the muscular metamers (myotomes) of the body should be ever borne in mind : the serial concentration of the neurons into ganglia corresponding with the serial segmentation of the muscles into myotomes. The second great group of bulbo-spinal neurons we have included under heading (2), *i.e.*, "*Other types*." It included a heterogeneous group of neurons in which hitherto chaos reigned supreme, but which, thanks to the aid given us by Golgi's methods of investigation, is gradually coming to be recognised as consisting of definite sub-types, which we shall now proceed to enumerate. Let us state at the outset that these neurons we are about to consider form a complex *intermediate* system in the bulbo-spinal cord, and that they have no peripheral connections in the strict sense of the word. The peripheral neurons are the earliest to be laid down in the economy, and belong to but two classes: (*a*) the *primary sensitive* (bipolar, epithelial), and (*b*) the *motor*, which directly innervates all the contractile (glandular and electric) tissues. The ones we are about to consider belong to an intermediate system, or as we may call it, an intercalated system (see fig. 4), interposed between the *primary* neuron on the sensitive side, and the primary neuron on the motor side; their connections are wholly intra or interneural: they have no peripheral connections. Of this intermediate system we recognise for convenience three types of cells which we shall call *association* or *connective* cells, *commissural* cells, and *projection* cells, the meanings of which will be rendered clearer as we proceed.

(1) *Association or connective neurons* (intrinsic) of the bulbo-spinal axis. The ganglionic chain of neurons is connected in *linear* series, as is shown by observation in all vertebrates and most invertebrates (whether such chain be *ventral* as in invertebrates; or *dorsal* as in vertebrates). And in the spinal cord of the higher vertebrates, as studied by Golgi's method, we find the actual nerve elements, which are specially concerned in affecting such inter-ganglionic connections, whether longitudinal or transverse. Those of the longitudinal system we had best call *associative or connective*, reserving the term *commissural* for those which connect the ganglia across. The longitudinal or associative neurons would thus serve to conduct nerve currents from one ganglion forwards or backwards, to *other ganglia of the same side*. *These cells* of the longitudinal system are very numerous in the spinal cord, and of moderate size—being smaller than the great moter cells of the cord-grey, whose motor fibres pass out *via* the anterior roots. They are scattered throughout the whole grey matter of the cord, some being found in the posterior cornu, others in the anterior, and others in the intermediate region. The majority of these lie in the deeper parts of the anterior cornu, and in the intermediate region. They have several branching protoplasmic processes, limited to the grey matter and a single nervous process which, passing outwards to the lateral column of its own side, turns off at a right angle, and runs

longitudinally; re-entering the grey matter either higher or lower down. These cells of the longitudinal tract systems might thus be aptly termed association cells, especially as in the brain such a term has been applied by Meynert to the fusiform cells in the deep layers of the cortex; and, indeed, to these cells, whose presence in the fœtal human cord has been shown by Leuhossek, that authors give the name of "Strangzellen." But these linear association cells send their fibres not only to the lateral column. Several of those which are situated in the posterior horn (of the kitten's cord) can be seen to send their nervous processes into the posterior column, where some of these fibres bifurcate in Y-fashion, one branch descending and the other ascending in the said column, while others simply turn off at right angles, and either ascend or descend as the case may be. When tangential sections are made of the cord so that slices are removed first from the peripheral parts of the posterior columns, and later, deeper parts (much the same as a cylindrical beam of wood might have strips of shavings taken off by a plane till the cross-section of the cylinder is no longer a circle, but semi-circular), we find that when we come to a region about half way between the posterior surface and the central canal of the cord, many such cells can be seen whose cell-body and protoplasmic processes lie in the posterior cornu; and whose axis-cylinder process, passing straight into the adjacent part of the posterior column, either turns and runs longitudinally upwards or downwards in that column, or else bifurcates into two branches, one of which runs upwards, and the other downwards. In successful specimens, we can further see that throughout their longitudinal course they give off five delicate collaterals at right angles, which re-enter the cord-grey and break up into a fine tuft of terminal fibrils like the jets of water in a fountain, and that ultimately the main fibre itself turns into and re-enters the cord-grey, giving rise to a similar but last tuft of fibrils. Each of these terminal tufts comes into close and intimate contact with the cell body and main protoplasmic processes of another cell situated higher or lower down the cord; thus establishing a longitudinal association in the strictest sense of the term. It would take us too long to follow this exposition further: suffice it to say that careful observation will reveal such association cells teeming throughout the cord in successfully prepared longitudinal sections of the fœtal or new-born kitten. Let us add that these intrinsic cells of the bulbo-spinal cord are not the only cells which exhibit such a phenomenon, viz., each cell by its axis-cylinder process and collaterals establishing relationship with a number of other cells in longitudinal series. It was shown by Ramon y Cajal, and future investigations have confirmed his discoveries, that a single posterior root fibre—which belongs to the bipolar cell on the posterior root ganglion—after it has entered the cord itself generally bifurcates in Y-fashion into an ascending and a descending branch (see fig. 4, sensitive bipolar cell and its branches as shown by the arrows), each of which gives off at right angles collaterals which enter into the cord and form terminal tufts, coming in close contact with various categories of the intrinsic cord-cells: *i.e.*, some of these collaterals come in direct contact with the large motor cells of the anterior cornu (long collaterals), while others (short collaterals) establish relationships with the intrinsic cells either of the posterior horn, or of Clarke's columns, or of the intermediate region. The spinal cord of the kitten, and of other higher vertebrates, generally thus show both plans of structure present side by side, viz., the genetically older fundamental reflex type (consisting of bipolar sensitive cell, its central distribution, and motor cell—*c.f.* fig. 3), together with the *later superadded and genetically younger* system of association cells which we have already described.

It might now be added that while the structures we have dealt with, viz., the bipolar extrinstic sensitive cell and the intrinsic association cells of the cord, maintain only associations in the longitudinal sense, other cells are also found in the spinal cord whose function seems to be to give out axis-cylinders which establish cross connections, *i.e.*, with the opposite side of the cord—much as the callosal fibres of the brain do. These may be described, therefore, as *commissural* cells. *Golgi* was the first to demonstrate their presence in the cord. They are cells of medium size, and have an axis-cylinder process which, after crossing to the opposite side, *via* the anterior commissure, pass into the adjacent white subtance of the anterior or lateral columns: while *Cajal* has shown (confirmed by Kölliker) that often such a crossed commissural fibre can be seen to divide into two branches, one ascending and the other descending, so that the connection established (on the opposite side) may be, presumably, with several neurons. Some of the cells of Clarke's column are of the commissural type, that is, give off a nerve fibre which crosses the anterior commissure, and thus passes to the opposite side, while others crossing over to the lateral column of the same side—forming, in so doing, a distinct fasciculus crossing the base of the posterior cornu—turn and run longitudinally in the cerebellar tract, mostly as ascending fibres, but some few as descending fibres also.

To complete the description of the intrinsic nerve cells in the bulbo-spinal cord, we must add another two groups (minor types) of cells, viz., *Golgi's* cells with short, branching axis-cylinder, and *Cajal's* pluricordonal cells. These are few in number. The first type of cell is found in the substantia gelatinosa Rolandi: it has a small cell body, and its nervous process, shortly after leaving the cell, branches at once into a loose and rather wide meshwork of terminal fibrils which terminate in the grey substance itself; embracing within its meshes the adjacent nerve cells of the posterior horn. It is to be looked upon as belonging to the class of *association cells*, the range of association however being more limited and solid, and limited to a cluster of cells of one segment alone; not spread out over several segments in series.

Cajal's pluricordonal cells comprise a few scattered stellate cells in the posterior cornu (in the embryo of the pigeon), giving off two or three axis-cylinder processes, one, it may be, to the posterior column, and the other to the lateral. These represent an embryonic phase, but whether they persist to adult life or not is doubtful.

The above completes briefly the description of the various kinds of cells present in the bulbo-spinal system,—both intrinsic and extrinsic (but not including cells which establish relations with the brain), and they may be recapitulated as follows:—

First.—The extrinsic sensitive bipolar cells—primary sensitive neurons.

Second.—The intrinsic bulbo-spinal cord cells.

- (*a*) *Motor cells* (including secretory, electric, &c.).
- (*b*) *Association cells* (longitudinal).
    - (1) Serial, interganglionic (with long axis-cylinder).
    - (2) Local, intra-ganglionic (Golgi's cells).
- (*c*) *Commissural cells.* (transverse.)

The above four groups of cells are necessary to constitute the vertebrate spinal cord, and we can see, how, with such a mechanism available—as in the spinal cord of the decapitated frog (Pflüger)—a variety of cord reflex actions can be elicited by appropriate stimuli, and how with such an intrinsic mechanism of associating, coordinating, and commissural neurons, certain reflex actions of a highly complex kind might be evoked such as would almost deserve to be called "purposive." I will not, stay, however, to correlate such reflex actions with the actual facts of the nerve mechanism, leaving that to the ingenuity of the reader, only stating *en passant* that even in a lowly organ like the spinal cord in vertebrates, the organisation of the nervous mechanism is such as to admit of a considerable variety in reflex actions.

In the higher vertebrates, especially mammals and man, the bulbo-spinal axis and its peripheral appendages form but the lowest portion of the nervous system (Hughlings Jackson's third or lowest level). For in them we have a new and marvellous nervous mechanism, placed *above* the bulbo-spinal system, the growth elaboration and importance of which have come to infinitely overshadow the lowly bulbo-spinal cord, viz., the encephalic system—cerebral and cerebellar.

What is the upward connection of the bulbo-spinal with the cerebral on the one hand, and the downward connection of the cerebral with the bulbo-spinal on the other? And how are we to look upon the cerebral system from the neurological standpoint? What is its morphological value, its significance, and how is it correlated to or united with psychical function and consciousness?

The Cerebral System.

The cerebral system is a new formation of neurons, a new organ developed in connection with the central projections of three great sensory systems, the *olfactory*, the *optic*, and the *fillet* radiations, the region of anatomical growth starting fundamentally from the first of these, the olfactory centres; the ingrowths of the optic radiations and fillet radiations soon following and adding to the complexity of the superstructure. The reason for its development at the anterior or cephalic end of the body is not far to seek. That was the part of the body which led in the movements of the animal; that was the part, therefore, to come first and most into contact with the external world. It would be the part first to receive stimuli; the leading part in locomotion, one, therefore, whose sensitiveness and reactiveness should be *most* developed, and which should, in short, constitute the guiding and initiative focus of movement which the rest of the body-segments followed in sequence and dependence. Hence it is in the cephalic region we find developed in animals the *antennæ* (feelers), the *optic* or *visual* organs, the *orientation*, sense organs (primary auditory vesicles), and the *olfactory* (and *gustatory*) apparatus. And that, therefore, is a region in which not only ganglionic concentration takes place (as in all segmented invertebrates, and in vertebrates), but a higher, more complex, growing and therefore more plastic, nervous mechanism is elaborated, which constitutes the encephalon in all craniate vertebrates. But this encephalon itself is composed of three great quasi-independent systems, viz., the *olfactory*, the *optic*, and the *cerebellar*, the relative predominance of one or more of these varying in different classes.

The main features, however, are concisely presented for each class in the following table:—

		Most.	*Less.*	*Least.*
Archi-encephala	(1) *Elasmobranch Fishes*	Olfactory.	Cerebellar.	Optic.
	(2) *Teleostean Fishes*	Optic.	Cerebellar.	Olfactory.
Palli-encephala	(3) *Amphibia*	Cortical.	Optic.	Cerebellar.
	(4) *Reptiles*	Cortical.	Optic.	Cerebellar.
	(5) *Birds*	Cortical.	Cerebellar.	Optic.
	(6) *Mammals*	Cortical.	Cerebellar.	Optic.

the growth of the *cortical* or *pallial* system increasing as we rise above the scale of fishes, and attaining its *maximum* in the higher mammals and *man;* such growth being correlated to the central projections of the three great sensory systems, the *olfactory*, the *optic*, the *bulbo-spinal* or *fillet radiations* within the growing pallium. When the conditions described, therefore, for the cortical type of nerve mechanism are realised, the foundations are laid for possibilities in nerve activity of an exceedingly complex kind, far above the level of the bulbo-spinal type, concerned, not only with bulbo-spinal sense projections, but with olfactory and optic sensations as well, and the development therewith of higher areas in which these sensations combine and cohere into higher (psychical) association; possibilities in nerve activity, therefore, which may grow up into the most elaborate forms of sensory and psychical activities,—mental acts of cognition and recognition, comparison, discrimination and judgment, initiation of movements and acts, spontaneity, conduct.

For consider for a moment what such cortical mechanism implies. The neurons of the cortex which have to do with the nerve currents of different (qualitative and quantitative) incoming sensory excitations are themselves aggregated into complex structures, into layers of cortical cells, into groupings and cell clusters, and into complexes of associated (unilateral or bilateral) clusters and centres as well; each such series of centres being built round its own distinctive sensorial platform. But over these again are built and elaborated higher strata and regions of nerve activity which have to do in an ascending scale with pre-conceptual and ultimately conceptual thought, the last of which leads into all the possibilities of mental life. We shall later in the paper deal with the question of psychogenesis and show how it corresponds to the neurological law according to which higher association systems develop as a later superstructure over the older ones. At present we shall proceed to consider in some detail the actual structural mechanism of the *pallium* (cortex), and especially try to ascertain what, if any, are the mechanisms or regions where the incoming sensory excitations are *received* by the cortical elements proper.

The Cortex.

Structure of the Cerebral Cortex and the organisation of its Neurons.—In this region, too, as in the spinal cord, the actual state of knowledge before Golgi entered the field may be fitly described in one word—chaos!—the labours of the most distinguished workers in this field having really thrown no light on the actual inter-connections and organisation of the cortical neurons; a territory in which fierce controversies raged over "fibrillary" and "granular" theories which were in vogue to explain connections or the absence of connections when facts were not forthcoming. The investigations of such workers as Gerlach, Wagner, Max Schultze, Stiede, Deiters, Krause, Kolliker, Exner, Meynert, Betz, Flechsig, Retzius and others too numerous to mention, after the best methods in vogue, teasing, maceration and isolation methods, preparations with carmine, hæmatoxylin, picro-carmine, the anilines, osmic acid, gold and silver-stains, all failed to throw light where most light was needed, not only as regards the connections of the nerve elements themselves, but also concerning those less known structures which were spoken of under the term neuroglia. We shall here concisely state what the actual knowledge consisted of as regards the nerve-elements, condensed and abstracted out of the works of the above mentioned authors:—

First.—The presence in the cortex of pyramidal cells, each with one process tapering off from its summit (apical process) and ending like a candle's flame, and a number of other smaller and shorter lateral and basal processes ramifying and gradually merging into and being lost in a ground substance.

Second.—The presence of axis-cylinder processes coming off only from the largest of these pyramidal cells (giant cells, Betz's cells), while the others were stated to have no visible axis cylinders at all.

Third.—The presence of a certain lamination in the cortex composed apparently of distinct elements superposed in four to six layers (more or less in number than these according to various authors). Thus, according to the enumeration of *Meynert* (Stricker's "Histology," vol. ii., 1872; and later in Meynert's "Psychiatry," vol. i., 1885), blindly followed by Huguenin, Luys and several others, these layers from without inwards were (in the frontal cortex) five in number, as follows:—

First layer "made up chiefly of the basis-substance and its connective tissue elements" (Meynert's "Psychiatry," 1885, p. 59), and apparently containing no nerve cells, but in which Exner later discovered a plexus of nerve fibres.

Second layer "sharply defined on its outer margin and consists of densely crowded pyramidal-shaped bodies which turn their apices towards the surface and measure about 10 m. in height. The internal boundary is also well marked, owing not so much to a change in the size of these nervous elements as to the lesser density of distribution. This is the layer of small pyramids" (Meynert, *loc. cit.*, p. 59-60).

Third layer composed of medium and large sized pyramids, the size of these generally increasing in the deeper part, though intermixed with a fair number of small pyramidal cells also. This is called the "formation of the cornu ammonis." The lowest and largest pyramidal cells of this layer, in addition to their apical and ramifying lateral base processes, have "one medium base process which runs in a diametrically opposite direction to the apex process, viz., towards the medullary substance" (Meynert, *Ibid.*).

Fourth layer composed of small cells "chiefly of a rounded-angular nature. This is called the granule-like or granular formation" (Meynert, *Ibid.*, p. 60).

Fifth layer of fusiform or spindle cells (the "claustral formation" of Meynert) interspersed especially in its superficial parts with rather large but short pyramids. "The further we proceed towards the white substance the more spindle-shaped nerve cells of about 30 μ. we find; these spindle-shaped cells sending processes towards the granule layer present the appearance of vertically compressed pyramids. But they never show anything in the least akin to a medium base process" (Meynert, *Ibid.*, p. 60).

Finally, as regards the cells of all these layers, little was known beyond the shape of the cell body and its main processes, but as regards the ultimate dispositions and endings of these processes, and as regards the ultimate path and terminations of most of the nerve fibres also within the cortex the most absolute ignorance prevailed, there being no facts to hand.

And thus to explain or imagine the dynamic connections of these nerve corpuscles, two theories were put forth, viz: (*a*) the doctrine of anastomosis between nerve cells by certain fine or coarse protoplasmic processes (Schroeder Van der Kolk's and Gerlach's plexus), and (*b*) the gradual merging and fusion of the other cell processes into a "basis substance, where the fibrillary and the granular structures are fused" (Rindfleisch, 1872); a two-headed hypothesis held almost up to the present day.

As regards the *white substance*, it was held to be composed of nerve fibres which fan-like radiated into bundles in the cortex, some of these entering the base of the large pyramidal cells, while as regards others—and these constituted the vast majority—their destinations and connections were wholly unknown, the generally accepted theory being the above-mentioned one, viz., that these nerve fibres (fibrils) gradually fused into and became continuous with, the basis substance, the processes of the adjacent nerve cells similarly passing into and getting fused with the same basis substance. Thus, when more recently (1882) a series of medullated nerve fibres were found by Exner in the superficial layer, which before that was stated to contain no nerve elements but only "basis substance, and its connective tissue elements" (Meynert), an explanation was put forth to the effect that these tangential (or arciform) fibres of Exner's plexus were continuous with the apical processes of the subjacent pyramidal cells, and were thus to be looked on as belonging to these cells. The above *resumé* concisely represents the body of facts and hypotheses which prevailed till 1886, when the publication of Golgi's great work "On the Minute Anatomy of the Central Organs of the Nervous System" appeared.

The Work of Golgi, &c.

Golgi, by his researches on the cerebral cortex and other nerve centres, may be said to have laid the foundations of modern neuro-histology, and pointed out to us a way by which we might penetrate into and understand more intimately than was possible before, the minutest and ultimate structural mechanisms and organisations of the cortical centres. He showed: *Firstly*, that the whole protoplasmic expanse and branchings of the nerve cells preserved their individuality up to their very terminations, and that such terminations existed. *Secondly*, that there was no anastomoses between the branching processes of adjacent cells) Van der Kolk's and Gerlach's theory). *Thirdly*, that there was no blending and fusion of any protoplasmic process with or into a basis substance (Rindfleisch's theory). *Fourthly*, each pyramidal cell in *all* the layers of the cortex, and similarly the *spindle and polymorphic elements* of the fifth layer, besides giving off the above-described apical and other protoplasmic process, gave off a process of different structure and appearance, the nervous or axis-cylinder process, and that each of these nervous processes gave off fibrils at right angles along its course. *Fifthly*, fine delicate fibrils (collaterals) which ramified into other fibrils of extreme delicacy, whose actual terminations seemed to constitute a most delicate plexus. *Sixthly*, that the cortical cells exhibited two sorts of axis-cylinder processes, viz.: (*a*) those with long axis-cylinders which preserved their individuality for a great length, and then passing into the white substance became a medullated nerve fibre; and (*b*) cells with a short axis-cylinder, which, rapidly branching and ramifying in the immediate neighbourhood, thus lost its individuality. It remains to add that *Golgi* thought—as a hypothesis pure and simple—that the cells of the former type, *i.e.*, with long axis-cylinder were motor, and those of the second type, *i.e.*, with short branching axis-cylinder, were sensory. Apart from the physiological theory which he put forth concerning the cells, it may be stated that *Golgi's* discoveries were confirmed in all their main particulars, the researches of subsequent workers—Fusari, Martinotti, Kölliker, Flechsig, Ramon y Cajal, Retzius and others having added certain details with some slight modifications to the work of the Italian master.

Material of Present Work.

In what follows, while acknowledging my indebtedness to the work of these distinguished neurologists, I must state that I have nowhere followed them blindly, and that while going over much of the ground independently, in the study of the embryonic brain of the *kitten*, as well as the brains of young and new-born kittens, rabbits, and rats, and in the further study of the adult brains of these animals, including the brain of the ox, I have been able to elucidate certain new, and I think, important, details which, especially in relation to the neuroglic elements, I have previously published in two papers during the past year.[1] The materials on which the present research is based, in so far as it concerns itself with the human cortex, consists of over a hundred human brains systematically prepared and investigated by the author during the past two years, with the main object of carrying on the inquiry into the higher regions of cortical structure, mechanism and organisation of the neurons in man. In this work, unlike that dealing with the brains of animals, I can quote no other observations besides my own, as I am unaware of any other work or publications on the subject; but the facts I am about to put forth are based on specimens which have been repeatedly shown to others, and notably at four scientific societies: (1) The Meeting of the British Medical Association at Newcastle, August, 1893; (2) The Leeds and West Riding Medico-Chirurgical Society, February, 1894; (3) The Annual Meeting of the Medico-Psychological Association of Great Britain and Ireland at Dublin, June, 1894; and (4) The British Medical Association at Bristol, 1894. I may also state that I shall be willing to show to any worker in neurology, the specimens which form the basis of my work. Having said thus far, I shall now proceed to give an outline of such facts as I have ascertained in this study of the human brain. I may state that, while in every case the method of staining after Golgi was adopted—slightly modified by the author[1] for the purpose of the present research on the human brain—other methods were also used, viz.: sublimate and alcohol fixation and hardening followed by staining in various anilines, especially toluidine-blue; the fresh method of freezing and staining with aniline blue-black after Bevan Lewis; and others with *hæmatoxylin*, gold chloride, Weigert's method for myelin, *congo-red*, picro-carmine, followed by aniline, gentian violet, and bacteriological stains followed by decolorisation; stains with acid rubin (Kultsckitzsky) and Biondi-Ehrlich triple stain, including Rosin's modification, Nissl's stain with methyline blue, and several others, too numerous to mention, some of these methods being for general purposes, and others to elucidate certain micro-chemical staining reactions of the cell protoplasm and the intra-protoplasmic structure of certain cells for purposes which will appear clearer later on.

---

[1] "The Neuroglia Elements in the Human Brain," *British Medical Journal*, August, 1893; and "On a System of Fibre Cells surrounding the Blood Vessels in the Brain of Man and Mammals, and its Physiological Significance," *Internat. Monatsch. für Anat. u. Physiolog.*, 1894

Cortical Lamination.

The various cortical layers are never strictly defined: elements of the one interpenetrate a little to regions of other layers. But allowing for this we recognise (taking the Rolandic region for our type) the following cortical layers, the names being mostly those of accepted nomenclature.

---

[1] "On a Modified Golgi's Method for the Study of the Human Brain," *British Medical Journal*, March, 1894.

(*a*) *The first or molecular layer*, containing but few nerve cells.

(*b*) The *second* layer of cells, sometimes fusiform, sometimes pyriform, and again, triangular or polygonal; interpersed with a large number of small pyramidal cells, but to be recognised as a distinct formation. One very prominent feature is the bifurcating thick upper part of the cell, looking like two unequal horns arising either out of the somewhat asymmetrical cell body, or from a short apical stump, in which latter case the cell is pyriform, globose, or fusiform. We recognise at least three types of cells concentrated into this layer, and excluding the interpenetrating small pyramids. Another noteworthy feature is the variability in form in these cells in various topographical areas of the cortex. This ambiguity of form might well justify us in calling this layer the ambiguous layer, a term which also serves the double purpose of recalling the two-horned nature of many of these cells (see fig. 5).

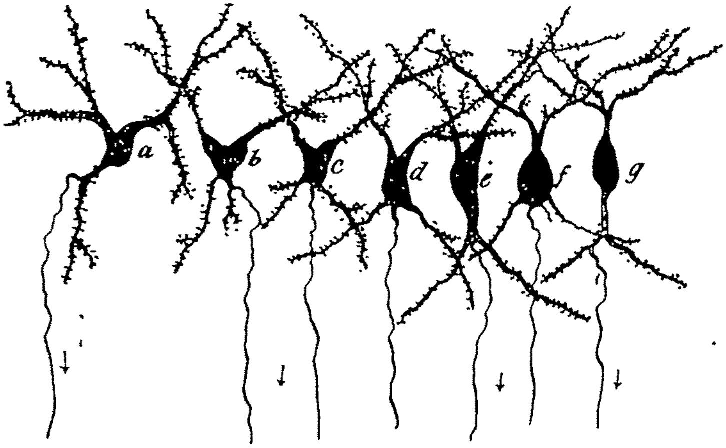


FIG. 5.

Seven cells of the second layer showing the main forms, viz:
a. and b. Asymmetical-bicornate.
g. Fusiform
f. Globose.
c. and d. Intermediate forms.
(From the brain of the ox.) (The *arrows* indicate the nerve fibres)

(*c*) *The third layer* of pyramidal cells with *long* apical process. We are inclined to divide the pyramidal cell layer into two classes, based on the length of the apical process; viz., the pyramidal cells with (*a*) short apical processes, and those (*b*) with long apical processes. The full anatomico-physiological reasons for this we shall give later on. The pyramidal cells with short apical processes are scattered throughout all the cortex below the molecular layer, and show no special gradation in size: none of their apical processes pass into the molecular layer: their terminations are always deeper. They may occur scattered, not only throughout the third, but the deeper polymorphic layer, and we look upon them as a different system of cells to the pyramidal cells with long apical process. For the latter always reach up to and end in the molecular layer; these are they which gradually increase in size of cell body from above downwards, besides presenting certain other characteristic features.

(*d*) The *fourth layer* of polymorphic elements in which the following are the main cell types: (1) Pyramidal cells with short apical processes. (2) Granule cells. Both these may interpenetrate a little distance into the superjacent layer. (3) Fusiform cells proper; with ascending axis-cylinder processes (first discovered by Martinotti), some of these cell bodies also occurring a little more superficially in the lower parts of the pyramidal layer (see fig. 7). (4) Fusiform cells with descending axis-cylinder processes, passing into the white substance. (5) Asymmetrical or oblique pyramidal cells, intermediate between the strictly fusiform and the pyramidal. (6) Inverted pyramidal cells (7) Cells with short branching axis-cylinder, and cell-body of polygonal shape. Many of these are shown in fig. 6.

Our investigations thus lead us to recognise the four following layers as fundamental to the Rolandic cortex in man, viz.: (1) Molecular; (2) Ambiguous; (3) Long pyramidal; (4) Polymorphic. And of the various cells entering into the constitution of these we recognised the following eight types.

(1) The pyramidal cell with long apical process, *i.e.*, reaching the molecular layer.

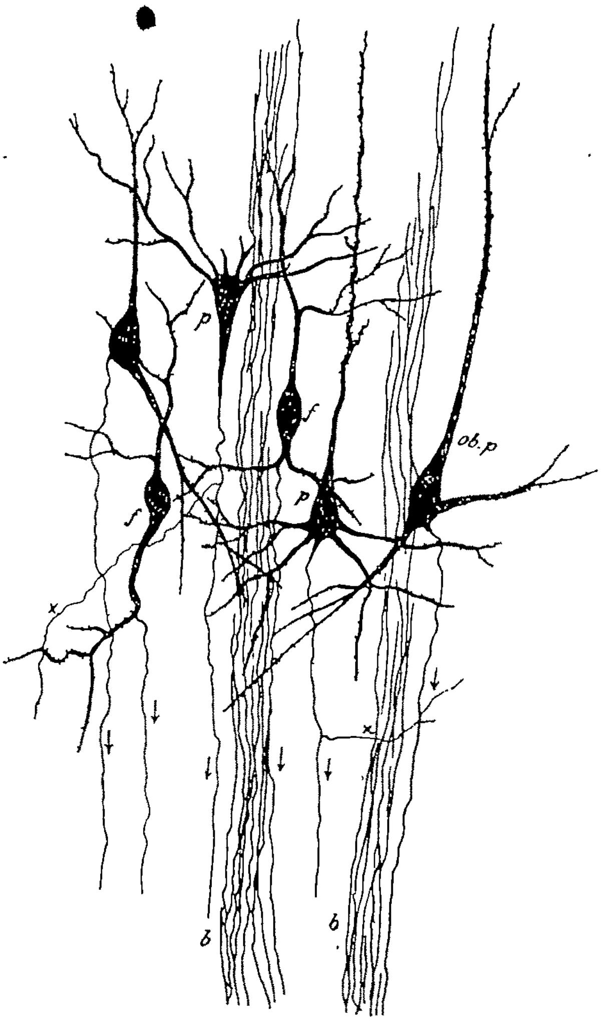


FIG. 6.

Nerve cells from the polymorphic layer (human cortex), stained by Golgi's method.

p. Pyramidal cells. (One of these is an *inverted* pyramidal cell.)
f. Fusiform cells. The arrows mark axis-cylinders.
ob.p. Oblique pyramidal cell.
x. Collaterals.
b. Bundles of nerve fibres.

(2) The pyramidal cell with short apical process, *i.e.*, not reaching the molecular layer.

(3) The ambiguous cell, whose sub-types may be asymmetrical-bicarnate, globose, fusiform, &c. (see fig. 5).

(4) The granule cell (fig. 18).

(5) The fusiform or triangular cell, with ascending axis-cylinder process.—Martinotti's cell (fig. 7).

(6) The fusiform cell, with descending axis-cylinder process (fig. 6).

(7) The oblique and the inverted pyramidal cell with descending axis-cylinder (fig 6).

(8) The polygonal cell with short branching axis-cylinder (Golgi's sensitive cell) (fig. 16).

(To these may be added certain cells of first layer [fusiform and triangular] described as present in the first layer by Cajal in which the differentiation into protoplasmic and axis-cylinder processes is obscure, and which are regarded by him as of embryonic nature.) We have not seen these in man.

Our investigations would thus lead us to the conclusion that there are no less than eight types of nerve-cells in the cerebral cortex, distinguished not only by their gross morphology, *i.e.*, shape of cell body and main processes, but what is even of more importance, by their anatomico-physiological connections, their topographical distribution, and—as we shall see later—also by certain differential characteristics of staining (micro-chemical difference), and of intra-protoplasmic structure. We shall now take up the individual layers in detail, pointing out the structure and anatomico-physiological relations of each.

First Layer.

First, the Molecular Layer.—This is the first layer. Its outermost or superficial region is formed of a system of neuroglia fibre cells, which from their characteristic form have been named by the author caudate cells (see fig. 8). These cells give rise to two distinct systems of fibres, viz.: (*a*) a tangential plexus of neuroglia fibres, radiating from the

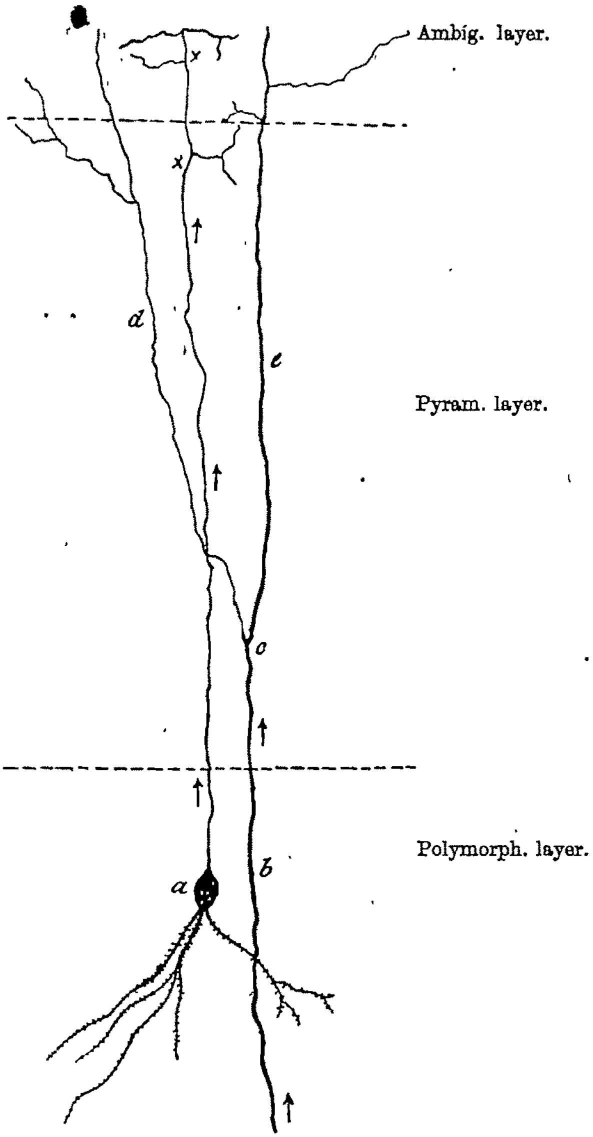


FIG. 7.

a. A fusiform nerve cell from the upper part of the polymorphic layer (human brain), with an ascending axis-cylinder (as shown by the arrows) passing up towards the surface (and giving off two collaterals at *x*).

b. A thick nerve-fibre ascending from the white matter, and giving off at *c* a fine branch *d*, while the main tube ascends *e*.

bases of these cells—surface condensation system; and (*b*) descending tufts of fibres, like a horse's tail, from the lower rounded apex of the cell, passing through the subjacent molecular layer; some of the longer of these fibres ending freely in the third layer. A study of fig. 9 will give one an accurate idea of these cells, both as regards the shape of the cell bodies, and the character and distribution of their fibre systems in the cortex. These cells were fully described and figured by the author, in a paper "On the Neuroglia Elements in the Human Brain," *British Medical Journal*, July, 1893. We may be permitted at this point to add certain minor details. I have stated of these caudate cells, that "they form a distinct feature in the first layer of the grey matter in specimens of human brain. The cell bodies are imbedded in the grey substance, and are the most superficial in position of cell elements—whether nerve or glia—in the cortex. The cell bodies have a rounded apex pointing downwards, and giving rise to a tail-like tuft of smooth fibres streaming into the deeper layers of the cortex. The wider and flatter base is directed towards the surface, and gives origin to an entirely different system of radiating tangential fibres (fig. 9). The individual fibres are extremely long, smooth-contoured, and of uniform calibre thoughout, of remarkably uniform thickness one with another, unbranched, slightly wavy in their course, which is, on the whole, almost rectilinear, and exhibiting here and there small sharp curves, and small angular bends, while sharp, transverse fractures are not infrequent. They form a fine felt-work of cortical (glia) fibres. . . . . . . None of the fibres anastomose, and none show any special vascular connections." (*British Medical Journal*, July 29, 1893.) Let us add, as a minor detail, that in addition to these, a few other glia cells are also found in the molecular layer, a little more deeply situated, and presenting certain forms intermediate between these caudate cells and the ordinary stellate fibre cells; two such transitional cells are given below in fig. 10, taken from the cortex near the bottom of a sulcus. These transitional cells unite the caudate cell to the stellate fibre cell which is found abundantly in the white matter, but only sparsely scattered in the grey matter of the cerebrum. Below this surface condensation system of neuroglia fibres we find the neuro-protoplasmic structures which, undefined and unresolved by the older methods of investigation, found apt expression in the term molecular substance, the actual structure and differentiations of which, however, we can now demonstrate, thanks to Golgi's method. About twelve different kinds of structures (glia and nervous) enter into its composition, and these are as follows :—(*a*) The descending neuroglia fibres from the caudate cells (figs. 8 and 9). (*b*) The cell bodies and fibre processes of certain other fibre cells (transitional cells (fig. 10). (*c*) Protoplasmic processes ascending and branching from the subjacent nerve cells of the ambiguous layer (fig. 5), and forming a dense and almost inextricable forest of granule-bearing branches and twigs. (*d*) Protoplasmic processes ascending and breaking out into terminal crown-like tufts from the *pyramidal cells with long apical processes* of the subjacent layers (fig. 11). (These are the pyramidal cells which, as we trace them downwards, increase in size as we go deeper into the cortex, ending with the largest or giant cells of Betz.) Cell bodies and (*e*) protoplasmic processes of certain intrinsic nerve cells of the molecular layer which have polygonal cell bodies, and a short axis-cylinder which branches and exhausts itself rapidly in the same layer (Golgi's sensitive cells). (*f*) Cell bodies and protoplasmic processes of certain protoplasmic glia cells, also found in this layer as in other layers of the cortex, and first recognised and described as a separate form of neuroglia cell by the author (in *British Medical Journal*, July, 1893); for further descriptions of which the original article must be consulted. Such a protoplasmic glia cell of the first layer is given in the accompanying woodcut (fig. 12). (*g*) *Nerve fibres* (axis cylinder processes, collaterals, and terminal ramifications) of various kinds, forming partly a medullated plexus of nerve fibres (Exner's plexus), and, intermixed with these, a rather larger number of naked axis-cylinders and their collaterals, and terminals unstained with Exner's osmic acid or Weigert's hæmatoxylin stains for myelin, but stained by Golgi's chromate of silver method (fig. 8). We have just stated that these nerve fibres, collaterals, and terminal ramifications are of various kinds. These include, first, thick nerve fibres, ascending from the white substance, and passing through the whole thickness of the polymorphic and pyramidal layers, to branch and end in the molecular and ambiguous layers chiefly. Such a fibre in the deeper part of its course is shown in fig. 7, from the thin occipital human

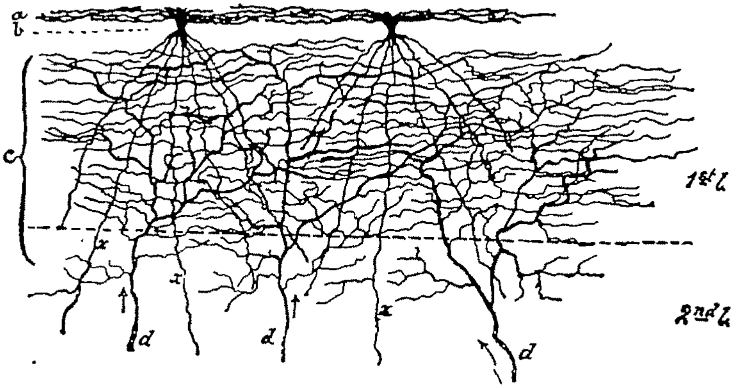


FIG. 8.

Human brain cortex, prepared by Golgi's method to show the various fibre elements of the 1st layer.

a. Tangential plexus of neuroglia fibres (surface condensation system).

b. Caudate fibre cell (of which two are given). These give off (*a*) the surface system of fibres and a descending tuft of fibres which penetrate deeply into the cortex (some of which terminate in the 2nd layer).

c. Plexus of nerve-fibres and collaterals.

d. Nerve-fibres ascending ↑ from the deeper parts of the cortex and branching and ending in a host of branches and collaterals in the superficial layer (constituting Exner's plexus). The fine collaterals and their terminations being devoid of myelin are stained only by Golgi's method; they do not appear in osmic or Weigert's preparations.

cortex, where it is sometimes possible to trace such a fibre from the white substance up to the second and the molecular layer; the further course and distribution of three of such gross fibres (*d*) being shown in fig. 8. These fibres have been aptly named "terminal fibres" by Cajal; their exact origin is obscure, as it is impossible to trace whence they come through the enormous extent of the centrum ovale. Assuming that they can come from one or more of three possible situations viz., : (*a*) other convolutions or lobes of the same side, or (*b*) from the opposite hemisphere, or (*c*) from more remote parts, *e.g.*, cerebellum, or bulbo-spinal

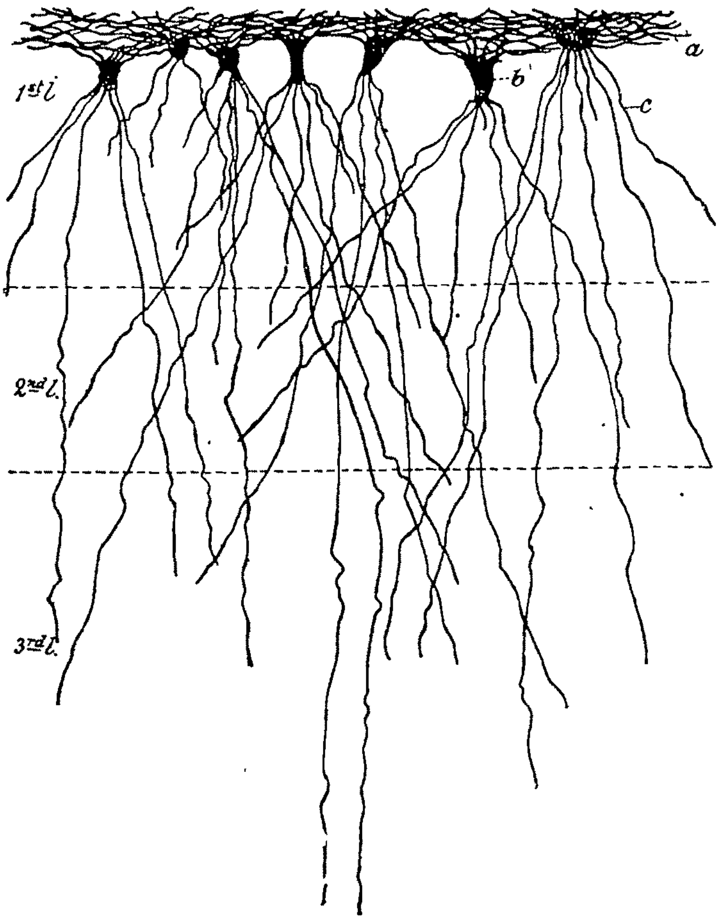


FIG. 9.

Seven caudate neuroglia fibre cells from the human brain cortex (1st layer).

a. Tangential fibre system.
b. Cell bodies.
c. Descending fibre system.

(The dotted line shows the limit between 1st and 2nd and the 2nd and 3rd layers.)

system, &c.; these fibres would be either long association fibres, commissural fibres, or projection fibres.

Besides these gross terminal (medullated) fibres (*g*), there are other fibres, viz.: (*h*) Fine terminal branches (fig. 7, *d*) and collaterals—both non-medullated—derived from the same thick fibres either as offshoots from the stem coming off lower down (fig. 7, *c*) or streaming off from the more peripheral branches in the molecular layer itself. (*i*) Ascending axis-cylinders from cells (fusiform, triangular, or polygonal) in the deeper parts of the cortex (fig. 7, *a*)—first shown to exist by Martinotti (1890). (*k*) Branching short axis-cylinders from local polygonal cells situated in the deepest strata of the molecular layer itself (Golgi's sensitive cells). (*l*) Ascending collaterals (few in number) from the axis-cylinders of the subjacent pyramidal cells. (*m*) Ascending collaterals (also few in number) from polygonal cells of Golgi's sensitive type, in or below the ambiguous layer

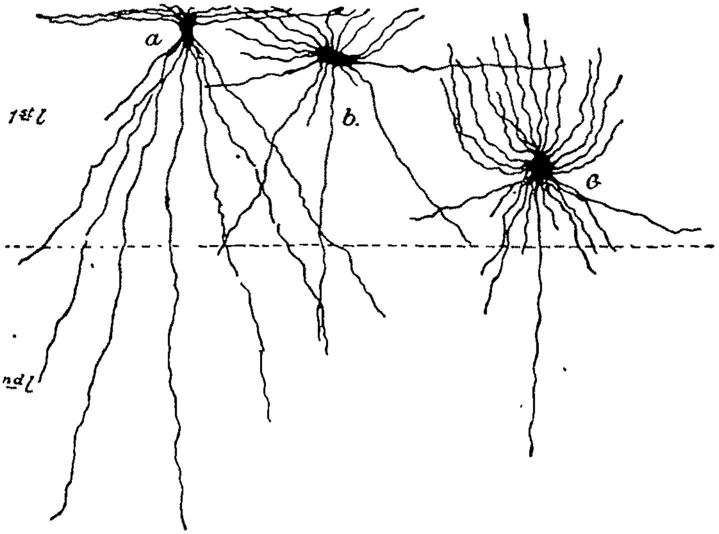


FIG. 10

Three neuroglia fibre cells from the cortex of the human brain (1st layer)

a. Caudate cell.

b and *c*. Transitional forms between caudate and stellate fibre-cells.

(The dotted line marks the limit between the 1st and 2nd cortical layers.)

itself. (*n*) Certain fusiform nerve cells in the upper part of the molecular layer, each with several ultimate nerve fibrils, mainly disposed horizontally (Cajal's cells). The above concisely represent the *dozen or more* structural elements (both nervous and neuroglia), pervading the molecular layer, the nature and connections of which (*i.e.*, whence they come and what their nature) have been so far determined. Is it possible to unravel the complexities of this structure, and to elucidate the various anatomico-physiological *sub-types* of connection, if such there be ? We are inclined to answer the question in the affirmative, basing ourselves on what is a well-known fact, that in various topographical regions of the cortex (*e.g.* Rolandic, occipital, hippocampal, cornual, &c.), sometimes nerve elements of one type, and sometimes of another preponderate, and thus allow us to study their connections more favourably. A careful study of this molecular layer is indeed absolutely necessary before we can make further progress, and a clear understanding of its nature will prove of the utmost value to us in entering into, and appreciating more intimately some of the deepest mysteries of cerebral action.

Pyramidal System.

Take first the whole system of what we have called "pyramidal cells with long apical processes," for these are the fundamental type in the Rolandic cortex, and are those which are conspicuous and easy to study. These gradually increase in size from above down, until when we come to about half way down the depth of the cortex, these cells are so large (Betz's cells) that they can, when stained, be distinctly seen with the naked eye, especially in toluidine preparations. The association of these pyramidal elements into clusters—cell nests of Betz—is also a favouring circumstance, as in well-stained Golgi specimens, not only of the human brain, but in those prepared from the sigmoid gyrus of the cat, their connections in the molecular layer can be studied more clearly. The prominent feature in the apical processes of all these cells is that the main stem proceeding from a cell, however deeply placed it might be, runs upwards towards the molecular layer, all the while preserving its thickness and integrity as a *stem*, while the lateral twigs that it—the stem—gives off along its course are few in number, short, and slender, and easily recognisable as twigs only. But when the apical stem arrives at the second layer it divides into three or four main parts, each of which rapidly spreads out like the crown of a palm tree, giving minor branches and twigs, which rapidly spread and end

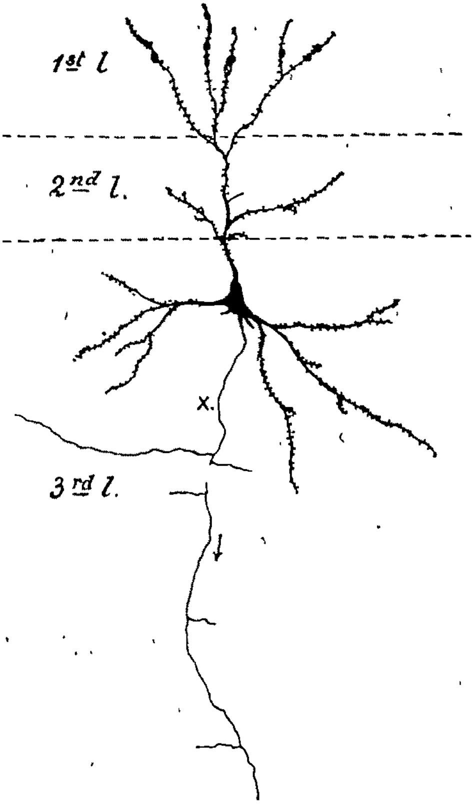


FIG. 11.

A small pyramidal nerve-cell of the human brain-cortex (3rd layer) showing commencing softening and swelling in the ultimate twigs of the apical process.

x. Axis-cylinder process giving off collaterals as it descends.

throughout the molecular layer, pervading the whole depth of that layer except the most peripheral part—the region of the tangential neuroglia fibres, and the caudate neuroglia cells. This is a practically universal feature, conspicuous in every specimen, and one which is especially easy of recognition in the cornu ammonis. Indeed, the cornu ammonis may be taken as the most valuable place to study (by Golgi's method) this system of pyramidal cells with long apical processes, and their connections in the molecular layer, for here the ambiguous layer of cells is absent —thus simplifying our study—while of the pyramidal cells only those with long apices are present, pressed down, as it were, into a well-condensed stratum of cell bodies about six or seven deep in man, while the whole of the cortex above (*i.e.*, stratum radiatum, stratum lacunosum, and stratum molecular, together) represents really the molecular layer of the Rolandic area. Here, then, we see the long pyramidal cell formation in its most typical form, and rather simplified—the principle we referred to before as one which might aid us in our investigations. That part of the pyramidal layer in fig. 13 of the cornu ammonis between x—x, shows such a condensation of the long pyramidal cells into a layer about six cells deep in man, about four cells deep in the monkey, and about three deep in the rabbit. My studies have been chiefly on the human and rabbit's brain; in a few cases on the kitten's brain also; and the chief conclusions concerning the molecular layer will be concisely stated. Taking the three main layers in this region, viz.: (*a*) *pyramidal* stratum, (*b*) sub-pyramidal stratum, and (*c*) *supra-pyramidal or molecular layer*; the last occupies about *seven-tenths* of the total thickness of the cortex, while in the Rolandic area its average relative thickness is about *one-tenth* of the total cortex. There is thus an enormous development of the molecular layer in the region of the cornu ammonis, rendering it a better field for study. The three strata mentioned in the text books, viz., ss. moleculare, lacunosum, and radiatum, we recognise as really sub-divisions of what is really one layer. We shall take these *seriatim* (see fig. 17 *infra*).

First, sub-stratum moleculare. This contains: (*a*) The most peripheral portions and endings of the ultimate protoplasmic twigs from the apical stem of the pyramidal cell. (*b*) A number of exceedingly delicate nerve fibres comprising the terminal collaterals of certain axis-cylinders, ascending from triangular and polygonal cells scattered sparsely in the sub-pyramidal (polymorphic) layer. These cells, therefore, are the same as Martinotti's cells already figured and described (fig. 7). (*c*) A few fine collaterals and terminals from cells in the stratum lacunosum—also sparsely scattered. These cells belong to Golgi's sensitive type. The protoplasmic processes of some of these cells are very long, some of the deeper protoplasmic processes reaching down to the polymorphic layer. (*d*) The cell bodies, protoplasmic process, and branching short axis-cylinders of fusiform and triangular nerve cells limited to this sub-layer very distinctly. (*e*) Lastly, some of the terminal ramifications of nerve fibres ascending from the white substance or alveus.

Second, Stratum Lacunosum.—This contains: (*a*) A large number of medullated fibres, and corresponds with Exner's plexus as shown by staining with Weigert's method. Intermixed with these are a large number of delicate (*b* and *c*) collaterals and terminals derived from two main sources, viz., those of nerve fibres ascending from the alveus, and those from the axis-cylinder branching and ending from *local* cells—triangular or polygonal—of the same layer. (*d*) Some of the large twigs and terminations of the apical process of the pyramidal cells.

Third, Stratum Radiatum.—Contains: (*a*) The main stem and its delicate and fine lateral protoplasmic offshoots. (*b*) Thick nerve fibres ascending to the more superficial parts, but giving off a few (two or three) branches in this layer as they pass through it. (*c*) Cell bodies, protoplasmic processes, and short branching axis-cylinders of a few triangular cells scattered in this layer, and belong to Golgi's sensitive type. (*d*) Collaterals from some cells of the sub-pyramidal layer: of these collaterals, some few *end* in this layer, but others *recurving downwards* reach the stratum of pyramidal cells, forming fine peri-cellular terminals which embrace the pyramidal cell bodies. We must add that the medullated nerve fibres of the stratum lacunosum, which we compared above to Exner's plexus, are derived mainly from cells situated in another part of the cornu ammonis, viz.: certain large pyramidal cells near the hilus of the fascia dentata—as was shown by Schäeffer (*Archiv f. mikrosc. Anat.*, Bd. 39, H. 4, 1892), and therefore these medullated fibres are the terminals of an association system, the cell bodies from which they are derived being in another part of the cornu ammonis, a little distance off. We shall say nothing of the non-nervous (*i.e.*, neuroglia) elements in this place except to repeat much of what we have already said,

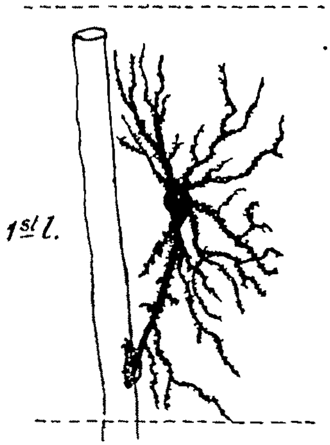


FIG. 12.

A protoplasmic glia cell from the *human* brain (1st layer of cortex) showing one expanded disc-like attachment to a vessel.

and without prolonging this exposition of the molecular layer further we shall lay down the following propositions, further evidence to which will accrue as we proceed.

Proposition First.—The molecular layer represents a region of the cortex where some nerve fibres coming from other parts of the cortex (*i.e.*, some association fibres), and similarly, we may add, commissural fibres coming from the opposite cortex (*viâ* the corpus callosum) have been shown by *Cajal* (in the new-born and young mouse's brain) to end, some of them in the molecular layer (while many others end in the deeper layers).

Do projection fibres end in the molecular layer? We have already stated that we look upon the cortex as a "new formation of neurons, a new organ developed in connection with the central projections of three great sensory systems, the olfactory, the optic, and the fillet radiations; the region of anatomical growth starting fundamentally from the first of these, the olfactory centres, the in-growths of the optic radiations and fillet radiations soon following and adding to the complexity of the superstructure." The existence of these central projections is an established fact, but what we seek at present to ascertain is where, in actually what layer or layers of the cortex, or in relation with what special neurons these sensory projection fibres do terminate? It need hardly be said that the question is of supreme importance.

Olfactory Projections.

It may be stated at the outset that we choose the region of the olfactory radiations or roots as the one most favourable for study, and that the summary of results obtained are given from a research of ours on this subject during the past two and a half years, yet unpublished. We used the brain of new-born and young kittens, and of rabbits for this purpose, and the following is a summary of our conclusions: (*a*) The olfactory radiations enter the brain by two obvious roots (external and internal), and a number of fine intermediate fasciculi; of these, some enter the anterior commissure and pass *viâ* it to the opposite side. These crossed fibres mainly come from the *inner root* as a distinct bundle, but many also come in smaller recurring fasciculi from the external root.

Leaving these crossed fibres, which are impossible to trace individually, we take up (*b*) the direct fibres. These comprise various sets, not all suited for our study. Some of these pass into the substantia innominata and head of the caudate nucleus, while others pass into undoubted cortex.

These cortical regions which receive the olfactory radiations are as follows: (1) The genual or pre-callosal part of the gyrus fornicatus; (2) the septem lucidum. Both these receive fibres directly from the inner root. (3) The inferior end of the hippocampal gyrus, and the gyrus uncinatus. Of these three regions we have chosen the first and the last, both undoubted cortex, and both places where the olfactory radiations can be followed satisfactorily.

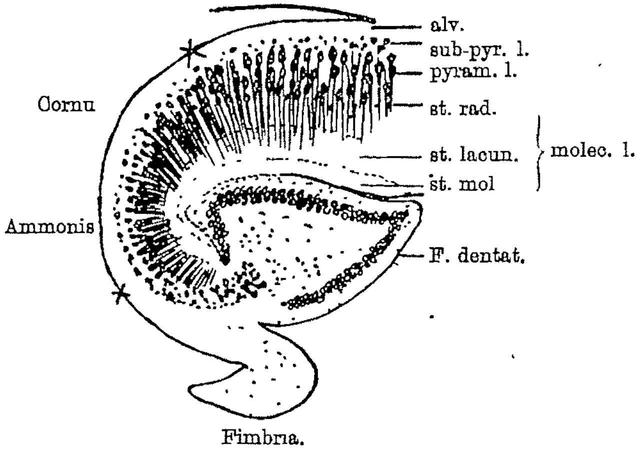


FIG. 18.

Cornu Ammonis, &c. (sketch of human). To show the regions studied in order to elucidate the connection of structures in the molecular layer.

First.—Pathway and terminations of the olfactory radiations in the *genual gyrus.* A considerable number of olfactory projection fibres from the inner root, and from the tract near its bifurcation, turn upwards at right angles, and leaving the root plunge into the cortical substance of the genual gyrus. In coronal sections made so as to include this root and the genual gyrus these fibres are seen as moderately thick medullated fibres, intermixed with a number also of finer collaterals, these being offshoots either of the same fibres or of other fibres going to the anterior commissure. The former can be traced *exactly in the situation of Exner's plexus, i.e.,* occupying the molecular layer; and in favourable sections can be seen to give off each an enormous number of collaterals, the fibre itself finally branching, ending in a plexus of terminal fibrils confined to the depths of the same molecular layer. The finer collaterals which come from the fibres going to the commissure also end freely, but only after a short course in the same molecular layer of the genual gyrus, as shown by staining with Golgi's method. In Weigert specimens — sections through both hemispheres of the kitten's brain—prepared from this region I have but seldom succeeded in tracing these derivatives; partly because the collaterals have no myelin sheath, and partly because it is easy to over decolorise these fasciculi. With the differentiation done in weak borax - ferricyanide, and continuously watched under the microscope—a tedious process—it is possible to demonstrate in preparations the medullated fibres spoken of, coming off from the inner olfactory; and the pathway of these for some distance along Exner's plexus, but no collaterals or terminal branches coming off from these can be seen, a fact which would seem to indicate that these are devoid of myelin.

Second.—Pathway and terminations of the olfactory radiations in the inferior hippocampal gyrus. These are derived from the conspicuous external root, and passing into the cortex over the inner and inferior surfaces of the hippocampal convolution can be traced often in fasciculi mainly running longitudinally but with minor oblique fasciculi, making the whole look like a loose meshwork of fasciculi, the meshes being elongated—throughout the whole extent, also, of the molecular layer. Some of these fibres in sections made vertically to the cortex, and parallel to their main direction, can be seen to give off, also, an enormous number of collaterals rapidly branching and intermixing in the most intimate association with the branched, tufted, and granulated protoplasmic twigs which ascend from certain elements in the subjacent layers. The same occurs in the genual gyrus with the olfactory radiations from the inner and trunk fibres. Having said thus far, we have now to inquire, what are the actual elements with which both these inner and outer olfactory radiations come in contact? And the answer is the same in both cases. They come into the most intimate relationship and contact with the apical protoplasmic tufts and twigs of pyramidal cells, the *region of such interconnection being in both cases the molecular layer*, and in each case the connection is with pyramidal cells *of the long apical process type*. Let us add—to complete our *resumé*—that in the inferior hippocampal region in question, in addition to these cells (both in the kitten and in the rabbit) we notice certain others, in a thin belt, and exactly corresponding in appearance and position to the ambiguous cells we have already spoken of and illustrated. The types present are either ovoid-globose or fusiform, or more asymmetrical and irregular (as in fig. 5). On the other hand, in the genual gyrus in the region corresponding to the internal olfactory distribution, these ambiguous cells are almost absent; a few scattered ones being here and there recognised. (Full descriptive details, with drawing, will be given in a memoir on this subject, in course of preparation.) Having said thus far, the chain of evidence is complete as regards the proposition we laid down before, and which, therefore, we must now legitimately re-state in the following form, as: *Proposition second*—The molecular layer represents a region of the cortex where sensory excitations coming from without, enter into relationship with cortical elements; and, *Proposition third*—That the said cortical elements are of two kinds, viz.: (*a*) Pyramidal cells with long apical processes, and (*b*) ambiguous cells. We therefore look upon these two systems of cells as the primary cortical neurons for the reception of sensory excitations. A word with regard to the optic radiations before concluding this part. The material we have utilised for the exact study of the intra-cortical terminations of the optic radiations is as yet too scanty on animals; and in the human brain the results obtained are as yet not conclusive. While confirming, in the main, Henschen's description of the distribution of these visual projection fibres to the calcarine cortex chiefly, we have quite recently been able to determine the exact situation or strata of the cortex where these fibres end, and what their connections might be. Specimens taken from a few (about seven) human brains certainly show an enormous number of medullated fibres, running chiefly antero-posteriorly, or, rather, in the *plane* in which these fibres spread, sheet-like, into each calcarine lamina, and most of the fibres in Exner's plexus here show a similar orientation—a suggestive, but by no means conclusive appearance. The plexus of Exner, here, is thick in the molecular, and in addition there is another below the ambiguous layer, and known as the strip of Vicq d'Azyr. With regard to the fillet radiations, the recent *resumé* of work done by Mott and others, and published by the former in the *Journal of Physiology* (1894), help us, unfortunately, but little in determining the exact distribution of the fillet fibres within the cortex. The question, we suspect, will have to be settled only approximately by degeneration methods (*e.g.*, the observation of myelin degeneration in the cortex by some method like that of Marchi's), before further conclusions can be drawn. Needless to say, however, all workers agree that the main distribution of the fillet fibres to the cortex is post-Rolandic, *i.e.*, to the ascending parietal and the occipito-angular and temporal regions, though some few of the fibres are pre-Rolandic in their distribution, but the exact question as to the stratum or strata of the cortex where these fillet fibres end, still awaits solution.

Ambiguous Cell-system.

We next come to a consideration of the second great layer of the cortex, viz., the ambiguous layer. The morphology of their cell bodies has already been described and illustrated in fig. 5. These cells and their grouping in the brain of the ox are seen in the next figure (fig. 14). The cell body is of various shapes—ovoid, fusiform, globose, triangular, with the base upwards, each angle of the base prolonged into a horn, and asymmetical. A single such cell from the brain of the new-born kitten is shown in another figure (fig. 15). The cells of this layer are well developed, especially in the hippocampal gyrus, and in the postero-inferior part of that gyrus they tend to assume enormous proportions. Here a special globose form predominates—as was first shown by Bevan Lewis in 1882 ("On the Comparative Structure of the Brain of Rodents"); indeed, of such size are they as to overshadow the bulk of the cell bodies of the subjacent pyramidal cells. We, for our own part, would add here further details, partly obtained by studying these by Golgi's method, and partly with various stains to note their staining reactions. Using the following stains, viz., Toluidine blue, Methyline blue, aniline blue, hæmatoxylin, &c., both after fixation and hardening with sublimate, and

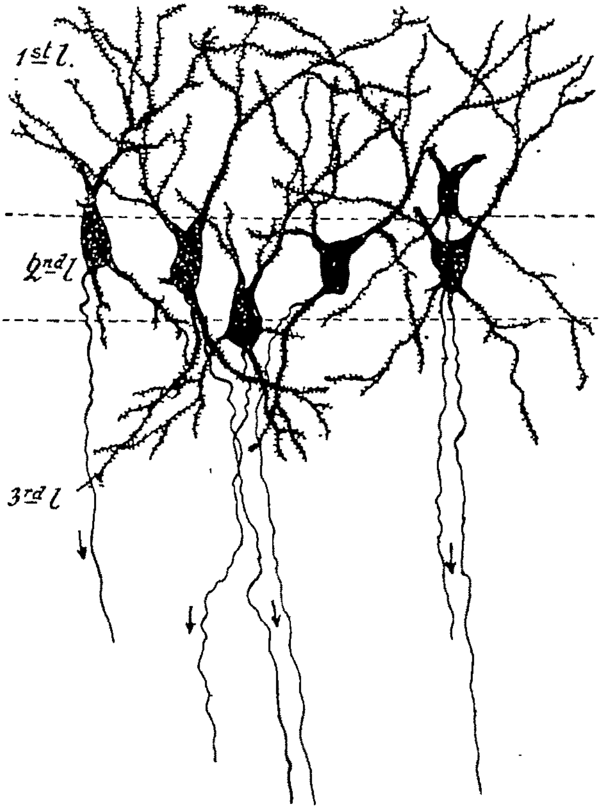


FIG. 14.

Brain Cortex of Ox, showing the cells of the 2nd layer.

1st l. Molecular layer.
2nd l. Ambiguous layer (with six cells).
3rd l. Third or pyramidal layer.

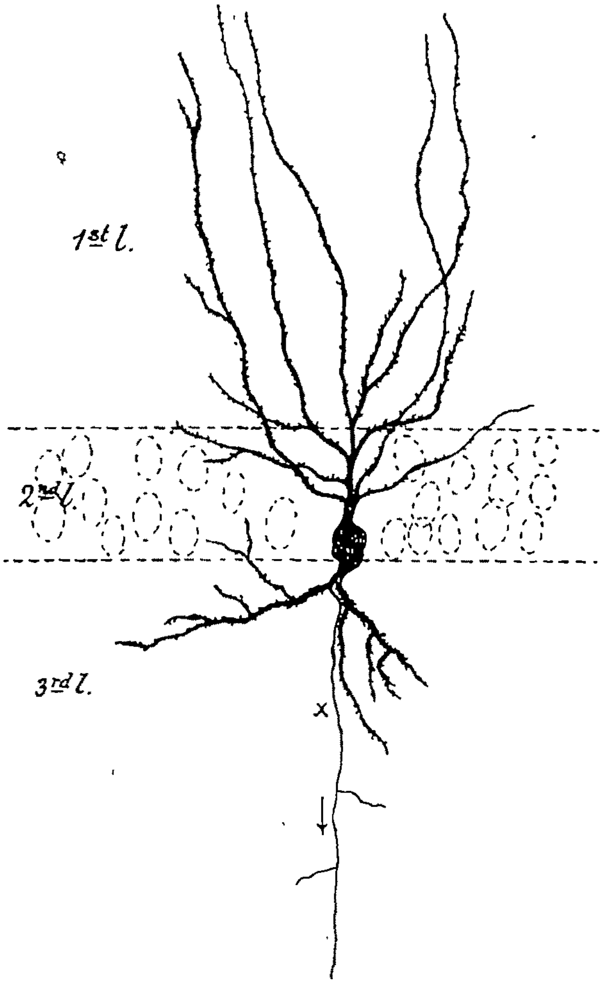


FIG. 15.

Nerve-cell from the 2nd layer of brain of new born *kitten.*

x. Axis-cylinder process, descending, and giving off two small collaterals.

1*st l.* Molecular layer.

2*nd l.* Ambiguous layer (one cell, and the dotted outlines of several cell bodies).

3*rd l.* Third or pyramidal layer.

also alcohol, followed by staining, we can state, with confidence, that these cells are distinctly chromophobic. They stain faintly, and their outlines are with difficulty visible, compared with that of the intensely stained pyramidal cell. The contrast is certainly striking; while the swollen bladder-like appearance of these cells cannot fail to make these objects of peculiar interest. Their cell-protoplasm also, is clearer—almost hyaline—and devoid of the chromatin granules, which, in the pyramidal cells, stain so intensely with all the above blues. Nissl's method, which is merely a refinement for the study of these granules, differentiates these two cell types for us in a similar way. With Golgi's method, they each show a distinct axis-cylinder, coming off sometimes from the middle of the lower border of the cell body, but more frequently it comes off obliquely, *i.e.*, from the side of the cell body, or from one of the basal protoplasmic processes not far off from the cell body (see figs.

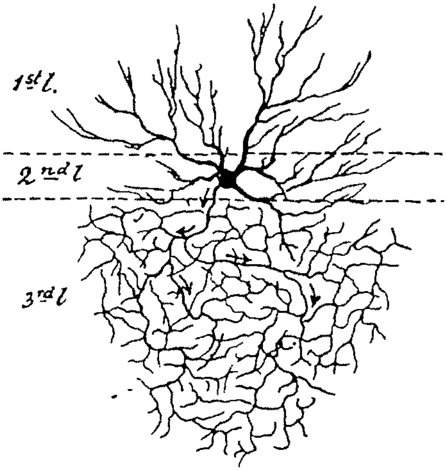


FIG. 16.

Cell from the cerebellum of the *kitten* (10 days old) showing protoplasmic processes and a branching axis-cylinder process whose twigs and collaterals ramify and get lost in the granule layer; the arrows showing the main stem and branches of the axis-cylinder.

1*st l.* Molecular layer.
2*nd l.* Layer of large cells.
3*rd l.* Granular layer.

14 and 15). These axis-cylinders descend downwards through the subjacent pyramidal layer, furnishing, as they go, three or four collaterals, but the ultimate destination of these fibres we cannot as yet positively state. They are exceedingly fine in calibre, and difficult to trace through the whole depth of cortex; some of them, at any rate, seem to end in the deeper parts of the cortex itself.

On the confines of the ambiguous layer—above and below it—appear other cells of a different type (Golgi's sensitive type), with an axis-cylinder process, which branches rapidly and rather closely into a wide-spread meshwork of terminal fibrils. These are cells which we have already described when dealing with the cornu ammonis, and the molecular layer (v. *infra*, fig. 17). It may be stated that while in the deeper part of the molecular layer, these cells have their network of fibrils mainly expanded out horizontally, the deeper lying cells (those *below* the ambiguous layer) have such fibril ramifications extending in all directions. These cells form a conspicuous feature in the cerebellum, and are there mostly chromophobic (fig. 16). In our specimens, too, of cortex prepared for the study of such cell reactions, these cells, whether above or below the ambiguous layer, invariably exhibited the same chromophobic character. Let us add, to complete the description of these cells, that wherever they appear—Rolandic area, cornu ammonis, or cerebellum—they are markedly chromophobic.

Long Pyramidal System.

We next pass on to consider the third great layer of the cortex, viz., the pyramidal layer or "*formation of the cornu ammonis*," one, the relations of which we shall try to understand with the help of the next woodcut (fig. 17).

The layer of pyramidal cells here, unlike that in other parts of the cortex, consists of but one kind of pyramidal cell, viz., that with long apical process. These cells are condensed here into a stratum of about six deep in man; the more superficial cell bodies being rather smaller and more ovoid in appearance, while the deeper ones are larger, and more pyramidal in shape.

Consider, first, the orientation of their protoplasmic processes. These are in two main regions, in two opposite directions, viz.: (*a*) The long apical stem system passing *upwards* through the stratum radiatum, and breaking up into fine, terminal, wavy branches in molecular and lacunar sub-layers, while furnishing also minor lateral branchlets in the radiate sub-layer; and (*b*) the short, basilar root system, of protoplasmic processes passing downwards into the deeper layer—like the fine, slender roots of a bulbous plant. This basilar system of protoplasmic processes ends mainly in this sub-pyramidal layer. In parts where the pyramidal cell body stratum is more diffuse, these relations are apt to be obscured, but here they can be studied to advantage, especially if together with human brains, we utilise those of the rabbit and the kitten to aid our investigation.

Taking, therefore, these two systems of protoplasmic processes so distinctively segregated into different strata, the question arises, What are the relationships of each of these systems. In other words, What are their anatomico-physiological connections with other neurons or systems of neurons? After a careful study of these regions, first by the methods of Golgi, supplemented next by the process of Weigert, and, lastly, the micro-chemical processes of coloring cell bodies already referred to, we find the following results obtained, which we will concisely summarise under three headings. *First* heading: All the pyramidal cells of the cornu ammonis are chromophilous. *Second*: Scattered throughout the other supra-pyramidal and infra-pyramidal strata are certain other cells, generally triangular or polygonal, and some few fusiform, which are markedly chromophobic; these present minor differences *inter se*, which, however, we cannot go into at present. *Third*: Weigert's method shows the following main systems of medullated fibres: those in the *alveus* or white substance (most marked), those in the *stratum lacunosum* (also marked), a few thick ascending ones, ascending from the alveus to the stratum lacunosum (some of them very well marked). The other substrata, viz., substratum *moleculare, radiatum*, and *sub-pyramidale,* show a few fine fibres, while the pyramidal stratum shows no fibres at all, except those passing *through* it from the alveus (white substance) upwards to the lacunosum (Exner's plexus). *Most* of the supra-pyramidal fibres are condensed into the lacunar region, forming a plexus which we can see to be homologous, with Exner's plexus of other cortical regions.

With *Golgi's* method now, we are able to unravel the connections of these fibre systems with the pyramidal cell, and we find that the most abundant nervo-protoplasmic connections occur in the lacunar sub-layer. To put things shortly, we have in this region the ascending and terminal branches of thick nerve fibres ascending from the white substance, and branching and ramifying into a thick plexus of collaterals and ultimate fibrils which intimately interlace with the abundant protoplasmic tufts and branches in that layer. One such fibre is shown in fig. 17 L. We may just pause here to turn back and compare this with what we saw before concerning the Rolandic or occipital or olfactory (hippocampal) cortex (fig. 8). The parallelism is complete. Medullated nerve fibres entering the cortex from other parts (as association commissural, or sensory projection fibres) pass into the molecular layer, branching and ramifying there to form Exner's plexus; the ultimate collaterals and terminals (non-medullated), from these coming into intimate contact with the apical terminals of the long pyramidal cells and the ambiguous cells. This latter class of cell being absent in the cornu ammonis, matters are further simplified and the nervo-protoplasmic mechanism easier to comprehend. While most of these collaterals and terminals end in the lacunar layer, a smaller number end in the superjacent stratum, and about an equally small number in the subjacent (radiate) layer. Thus the nerve fibre which enters the cortex of the cornu ammonis and proceeds to Exner's plexus, establishes connections with the whole anatomical expanse of the apical system, *i.e.*, with the terminal branches and tufts in the lacunar and superjacent layer, and with the delicate lateral branchlets of the apical stem in the radiate layer. To complete the description let us add that besides these thick ascending fibres, there are certain other fine ascending fibres which also come into the lacunar region. These are the ascending axis-cylinder processes of cells in the sub-pyramidal region (Martinotti's cells, see fig. 17 M, and compare with it fig. 7, where the same two kinds of fibres are shown from the occipital cortex). These axis-cylinders, ascending upwards, break up into a fine plexus of terminal fibrils mainly in the lacunar region, too, thus entering themselves into the most intimate connection with the apical protoplasmic tufts of the pyramidal cells. *What are the connections of the basilar protoplasmic system* of the same pyramidal cells? These are also possible to unravel. In this region we find a system of fine nerve fibres ascending from the alveus, and rapidly subdividing into collaterals and terminals in contact with the basilar system of protoplasmic processes. Three of these short ascending fibres are shown in fig. 17 Y. Their ultimate terminals are non-medullated, and they cross and interlace into a nervo-protoplasmic plexus with the basilar-protoplasmic processes of the pyramidal cell. We thus see the long pyramidal cell enter into relationship by its two poles, so to speak, with two sets of nerve fibres coming from elsewhere, *viâ* the white substance, viz., by its apical system in the molecular region (region of Exner's plexus), and by its basilar system in the sub-pyramidal region. These connections, therefore, may be termed extrinsic, to distinguish them from certain intrinsic relationships which these pyramidal cells enter into with adjacent cells. Such cells are the chromophobic ones we have mentioned as scattered throughout the supra- and sub-pyramidal regions of the cornu ammonis. Studied by the aid of Golgi's method we find that they all resolve themselves into Golgi's type of cell, with short branching axis-cylinder. We give drawings of five of these in fig. 17; one in the most superficial sub-layer (*c*), the arrows marking its axis-cylinder process; one in the lacunar layer (G‴); one in the radiate layer (G″); and two in the sub-pyramidal layer (G and G′). Each of these, as well as the remaining cell (Martinotti's cell) in the sub-pyramidal region, is shortly

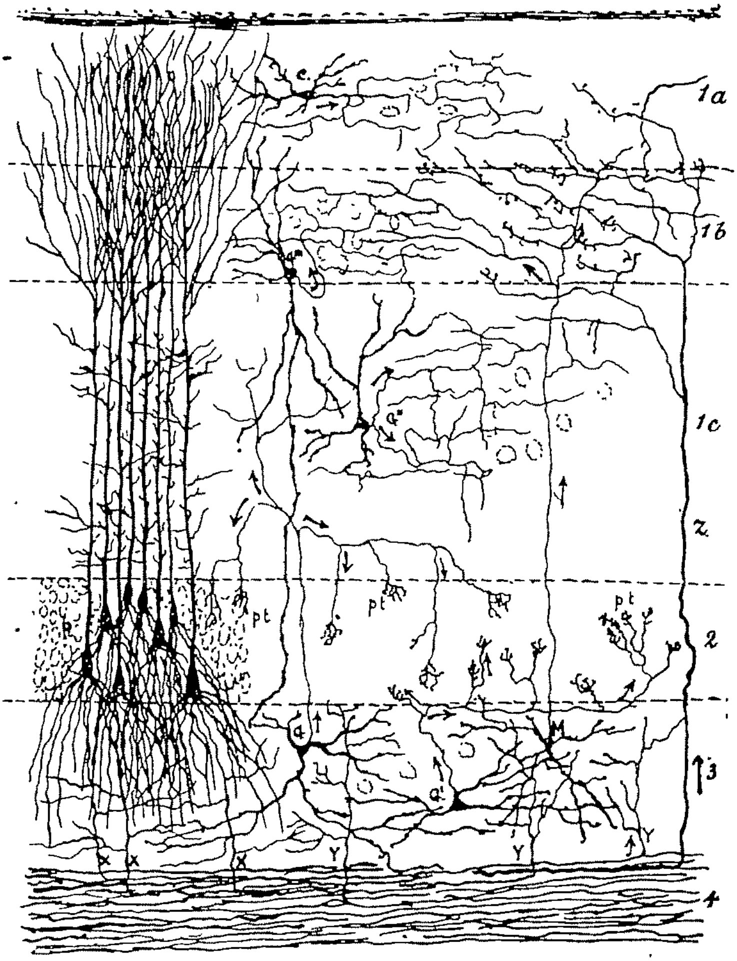


FIG. 17.

Cortex of cornu ammonis (human): prepared from several specimens stained with Golgi's method, and some with Weigert's method. 1 *a*. Substratum moleculare. 1 *b*. Substratum lacunosum. 1 *c*. Substratum radiatum. 2. Stratum pyramidal. 3. Sub-pyramidal layer. 4. Alveus.

P. Pyramidal cells in a stratum about six deep, the deeper cells being larger.

G. Golgi's cell, with axis-cylinder passing into stratum radiatum and dividing into collaterals of two sorts, some ending in that layer, others recurving and descending, and ending in the pyramidal stratum in terminal pericellular tufts (p.t.).

G'. Golgi's cells, with axis-cylinder passing upwards into the pyramidal stratum, and giving off several *upcurving and ascending* collaterals, ending in the pyramidal stratum, also in terminal pericellular tufts (p.t.).

M. A cell with ascending axis-cylinder passing into and ending in Exner's plexus (st lacunosum).

G″. Golgi's cell of stratum radiatum, its axis-cylinder branching and ending locally.

G‴. Golgi's cell of stratum lacunosum, with axis-cylinder also branching and ending locally.

C Cajal's cell of substratum moleculare; axis-cylinder branching and ending locally

X. Axis-cylinders descending from the pyramidal cells into the alveus or white substance, and giving off each a few (three or four) collaterals in the sub-pyramidal region.

Y. Short nerve fibres ascending from the white substance, and ending by branching freely in the sub-pyramidal region.

Z. A long nerve fibre ascending from the white substance, and passing through the pyramidal and radiate layer, to branch and end freely in the lacunar and molecular layers.

described in the notes attached to the woodcut. The pyramidal cell is thus a double mechanism, the recipient of nerve excitations which may reach either its apical system, or its basilar system, and each in two ways: (*a*) from extrinsic neurons, and (*b*) from intrinsic (adjacent) neurons. Let us add that a third factor is present which brings the cell body itself into similar nervo-protoplasmic relations. Certain cells of the sub-pyramidal region (fig. 17 G and G′) have axis-cylinder, which, passing upwards, give off' collaterals of two sorts: (*a*) descending and recurving collaterals (of G); and (*b*) upcurving and ascending collaterals of G′. Each such collateral ends in a tuft of terminals closely applied around the individual cell bodies of the pyramids. So that each such terminal pericellular tuft (p.t. fig. 17) can carry a nervous excitation from either of the cells G or G′, to the cell-bodies of the pyramidal cells. We must, therefore, extend our proposition; and now state that the pyramidal cell is thus a treble mechanism, the recipient of nerve excitation which may reach either its apical system, its basilar system, or its cell body; in the latter case by a specially differentiated pericellular tuft of fibrils derived from certain intrinsic cells in the immediate neighbourhood. These special cells we have not been able to find in other parts of the cortex (Rolandic, occipital, hippocampal or fornicati gyri, &c.), the only other situation in which similar ones occur being the cortex of the fascia dentata.

Regional Variation.

Before passing away from this part of the subject, we must take note of two or three things which are conspicuous by their *absence* from the cornu ammonis; and these are shortly—

(*a*) The ambiguous cell.

(*b*) The short pyramidal cell (which we shall study later).

(*c*) The granule cell.

(*d*) The oblique pyramidal cell and the fusiform or spindle cell.

We shall later have occasion to show that the *short pyramidal* cell, the *spindle* cell, and the intermediate form between these two, viz., the *oblique pyramidal* cell, belong to one great system. We may, therefore, re-state the position thus.

The cell systems *absent* from the formation of the cornu ammonis are three in number, viz., the ambiguous cell, the granule cell, and the short pyramidal and spindle formations; while per contra the intrinsic cells with short branching axis-cylinders (Golgi's sensitive type) are present in abundance in four strata, the cells of each coming into relationship with a separate part of the pyramidal cell: thus in fig. 17, the cell in the molecular sub-stratum (*c*) has an axis-cylinder and collaterals which interlace amongst the most distal endings of the apical system: the cell in the lacunar-layer (G‴) establishes connection with the coarser twigs, the third cell (G″) similarly is in relation by its collaterals with the main stem and delicate lateral offshoots of the apical stem; finally, a fourth category of cells (G and G′) establish connections (by means of terminal pericellular tufts) with the cell bodies themselves of these elements.

Finally, what of the nerve fibres *issuing* from these pyramidal cells? These have been shown by Golgi to leave the cell body at its base, and passing downwards as a nerve-fibre through the subjacent layer (see fig. 17, x) to enter into the white substance ; giving off at right angles in its course through the sub-pyramidal layer three or four delicate collaterals which ramify a short distance, and then end in the depths of same layer. This finishes the short but rather tedious account of the long pyramidal cell in the cornu ammonis.

Returning now to the cortex of the Rolandic area our way is prepared for a clearer conception of these cells, which we must now take up.

Cortical Sensory Mechanisms.

We have seen that wherever we could trace the intra-cortical terminations of sensory fibres—projected into the cortex either from the olfactory radiations, or probably also from the optic radiations—we found that their cortical terminus was in the molecular layer chiefly ; and, further, that they came into relation with the apical expansions of two kinds of cells, viz., the long pyramidal and the ambiguous, and that, therefore, these cell systems must be looked upon as the primary cortical neurons for the reception of sensory excitations. (Proposition third, *ante*, p. 593). If it is permitted to argue from analogy, aided by the fact that the fillet radiations end mainly in the cortex of the brain which is behind the fissure of Rolando—viz. : the ascending parietal, paracentral lobule, angular and occipito-temporal—though some of the fibres are also pre-Rolandic as well—it would be reasonable to infer that these fibres similarly end in the cortex like other sensory projection fibres, viz., entering into the cortex from the white substance, and spreading out superficially in the molecular layer as Exner's plexus. Slight faradic stimulations of the Rolandic cortex superficially may (as we know) produce in a patient certain sensations of a tactile or cutaneous nature which he can refer to a peripheral part (limb, &c.) This has been rendered probable by the case published by Dr. Ransome (BRAIN, liv.), and a case of Dana (*Medical Record,* May, 1893), of faradic stimulation of the Rolandic cortex. Further, in Jacksonian epilepsy of a slight kind the first distinct feature is the *aura*, which in this case is referred to, or starts from a certain definite part according to the site of the cortical mischief. Thus, if in the hand area, the aura consists in a sensation felt in the thumb or fingers. As the discharge spreads the next feature appears, viz., the tonic spasm of the hand, then (perhaps) a few clonic movements, and all is over. The feeling or consciousness of such a sensation on the psychical side must mean that there is a nervous excitation in the primary cortical neurons which in health habitually receive the sensory excitations from the hand *viâ* the roots of the spinal cord; the cortical neurons in question being, as we have every reason to believe, the ambiguous cells, and the long pyramidal cells of the "hand area" in the Rolandic cortex. That this Rolandic cortex is possessed of sensory endowments, partly tactile and partly kinæsthetic, is further proved on experimental and pathological grounds, such evidence having slowly accumulated ever since the time of Hitzig's earliest work in 1870. Without enumerating the cases or literature of the subject—already admirably done in Dr. Mott's paper, "On the Sensory-motor Functions of the Central Convolutions" (*Journal of Physiology*, 1893)—it may now be stated that lesions of the Rolandic area are accompanied by distinct loss of tactile and muscular sensibility: in other words, that the Rolandic cortex is the recipient of sensory excitations coming to the cortex *viâ* the spinal sensory roots and the fillet. We may, therefore, conclude this, the first part of our research, with the following propositions.

Conclusions of Part I.

(*a*) That the cortical terminus of sensory incoming excitations from other parts (whether olfactory, optic, or fillet radiations) is by medullated fibres which, entering the cortical substance, spread and ramify as medullated fibres in the molecular layer chiefly (Exner's plexus).

(*b*) That while the main fibre and its branches are medullated (insulated), its fine collaterals and terminals in the molecular layer *are naked* (*i.e.*, devoid of myelin).

(*c*) That these collaterals and terminals (fibrils), come into the most intimate contact with the enormous expanse of protoplasmic branches and twigs which ramify in the same layer.

(*d*) That this protoplasmic ramification is derived from two systems of cortical neurons, viz.: First, the long apical expansions of the pyramidal cells, and second the bifurcate expansions of the ambiguous cells.

(*e*) That these, therefore, are the first cortical cells which receive incoming sensory excitations; in other words, that these are the first sensory cells of the cortex.

PART II.

Having thus established the general doctrine of the primary cortical neurons which have to do with the reception of sensory excitations coming from without, it remains to push the inquiry further regarding certain details. We have seen that the main bulk and body of these sensory excitations reach the molecular layer where they break upon (*a*) the peripheral tufts of the apical systems of the long pyramidal cells; (*b*) the bifurcating horns and tufts of the ambiguous cells, the whole depth of the molecular layer being pervaded by the nervo-protoplasmic plexus which is the seat of such activity. We have also noticed how in the absence of the ambiguous cell—viz., in the thin cortex of the cornu ammonis—the same nervo-protoplasmic plexus exists, and that its most abundant development is at the lacunar stratum level. Contrasted with this rich nervo-protoplasmic apparatus of the apical system, we noticed also in the cornu ammonis that the basilar processes or root system of the pyramidal cells also occurred in connection with the less extensive nervous fibre-plexus mainly derived from *short* fibres ascending from the alveus, and ending in terminal arborisations in that region, while the *long* fibres which ascended to the lacunar layer contributed to the formation of the apical nervo-protoplasmic apparatus. We thus have two regions or strata, each the seat of a distinct organisation. The ambiguous cell is not present here, and our exposition therefore of its connections must now be detailed. Part of its connection, however, we have already shown, viz., that by its bifurcate apical system it too, enters into the constitution of the superficial nervo-protoplasmic plexus. Has it too a root system like the pyramidal cell, and if so what is its organisation ? Our investigations seem to show that the ambiguous cells too, have a basilar nervo-protoplasmic system like the pyramidal cells, and that such can be undoubtedly demonstrated where the ambiguous cells form a well-marked stratum.

Organisation of Visual Area.

Such a condition occurs in man in the cuneate region of the occipital lobe, in the brain of the rabbit in the *upper part of the retro-limbic area* (that called by Bevan Lewis the modified upper limbic area). On the mesial aspect of the occipital pole in the rabbit's brain there is a fossa-like indentation of the cortex which is applied like a cap over the mesencephalon and part of the cerebellum. This spoon-shaped area of cortex is indented by a faint furrow running backwards almost to a point a little *below* the occipital tip; we regard it as the homologue of the anterior part of the calcarine fissure. The part of the cortex *above* this fissure is the area we propose to investigate. On making a section of the cortex here we notice a striking feature, visible to the naked eye, and that is the presence of a fine white line in the cortex horizontally placed in the cortical layers about half way down their depth, thus dividing the grey cortex into an upper and a lower stripe. In the human brain this stripe is present in the calcarine cortex only (*i.e.*, in the depth of the calcarine fissure, and at its lips), as was shown by *Gennari*, and here in the rabbit it can also be recognised as a fine white line. It may be called *Gennari's* stripe, but has also received the name of Vicq D'Azyr or Baillarger's stripe. The following are the layers and their thicknesses in this part of the rabbit's brain.

(*a*) *Molecular layer*	$\frac{2}{8}$	thickness of	whole cortex.
(*b*) *Ambiguous layer*	$\frac{1}{8}$	,,	,,
(*c*) *Gennari's stripe* (*d*) *Pyramidal layer*	$\frac{4}{8}$	,,	,,
(*e*) *Polymorphic layer*	$\frac{1}{8}$	,,	,,

The total *thickness* of *Gennari's* stripe is about $\frac{1}{6}$ of the total cortex, and it lies immediately beneath the layer of ambiguous cells. These ambiguous cells form a stratum separating, therefore, the molecular layer (Exner's plexus) from Gennari's stripe, and these cells in this situation demand further study.

We find that in this region these cells form a well-marked and dense stratum, about six or seven cell bodies deep, the upper border of this stratum being especially well-marked, while the lower border of the stratum is less well-defined owing to its looser structure and the presence of straggling cells. The whole nature and character of cells is as already described for the brain of the ox or cat, and we will not stay to particularise them further. We need only add that while their cell bodies are mainly ovoid or fusiform, there are present both apical and basal processes. The apical processes bifurcate, and rapidly branch and end in the superjacent molecular layer, entering into the formation of the nervo-protoplasmic plexus in that layer, to which the apical processes of the long pyramidal cells also contribute. But it is the basal system of processes which here claim our attention, for these also branch and sub-divide forming a plexus, to which the pyramidal cells contribute only a few lateral branchlets of the main apical stem as it passes through to reach the molecular layer. This region is the place of *Gennari's* stripe, and we may add that Gennari's stripe (as we can see in specimens prepared by either Golgi's method for the rabbit's brain, or Weigert's method for the human, rabbit, and kitten's brain), is composed of a stratum of (medullated) nerve fibres, naked fibrils and collaterals which in this situation mainly run tangentially throughout its whole extent. We regard them as derived mainly from the nerve fibres which constitute the optic radiations of Gratiolet. The details of our research on this point will be embodied in a separate paper, but here we may state that examination by both the silver and Weigert-Pal method show us that the bundle of the optic radiations as it passes backwards from the most posterior end of the internal capsule, spreads out fan-wise towards the occipital pole (as seen in low powers), and then penetrates into the cortex of the area where Gennari's stripe is seen. The nerve fibres enter the cortex from the white substance below, and passing upwards as thick medullated fibres can be traced mostly into Gennari's stripe, and others still further up into the molecular layer. These are incoming fibres—viz.,coming into the cortex from the optic radiations, and terminate in the cortex by free plexiform arborisations of wide extent. Both the ambiguous cells and the pyramidal cells can be seen giving off each its own descending axis-cylinder with collaterals. Those of the ambiguous cells are especially *delicate and fine*, and cannot be confounded with these thicker and coarser fibres which ascend in the cortex, giving off branches and getting thinner as they ascend. In fortunate preparations the latter can be traced as they give off large coarse branches which turn off and run on for enormous distances in Gennari's stripe, and from these coarse branches numerous delicate collaterals are given off all throughout the depth of Gennari's stripe, the vast majority of these ultimate fibrils and collaterals being disposed horizontally. Some of these coarse fibres pierce the ambiguous layer, and reaching the superjacent molecular stratum, branch and spread out there in a rich profusion of collaterals and terminals. It would be impossible to estimate the exact richness in fibrils and collaterals of both these plexuses, and the present writer, after a careful study of Golgi's specimens, comes to the conclusion that the two regions are about equally rich in nerve-fibres and collaterals. We have thus a very instructive nervous mechanism revealed in these two regions. We have *first* the nervo-protoplasmic plexus of the molecular layer—with which our previous studies have made us familiar—a structure wherein the incoming sensory excitations reach the apical expansions of the pyramidal and the ambiguous cells; and *second* the *new* nervo-protoplasmic plexus in Gennari's stripe, subjacent to the ambiguous layer (see fig. 22), where the incoming sensory excitations enter into relationship with the basilar expansions of the ambiguous cells, and to some extent with the lateral branchlets also of the pyramidal apical processes which pass through it. And our parallelism is now complete, for where we can study the pyramidal or the ambiguous elements in well condensed strata we find each of them possessed of two systems, viz., (*a*) a well marked and ramified apical system, and (*b*) a less marked basilar system, and that each of these protoplasmic systems come into connection with a system of nerve fibres and terminals, forming in the cortex *three* primary nervo-protoplasmic plexuses whereby incoming sensory excitations can affect either of these two kinds of cells. These three nervo-protoplasmic plexuses are as follows:—

(*a*) The nervo-protoplasmic plexus in the first layer (molecular).

(*b*) The nervo-protoplasmic plexus in the third layer (Gennari's stripe).

(*c*) The nervo-protoplasmic plexus in the sub-pyramidal (polymorphic) layer.

or in other words we have—

(*a*) The nervo-protoplasmic plexus common to both long pyramidal cells and ambiguous cells (molecular layer).

(*b*) The special basilar nervo-protoplasmic plexus beneath the ambiguous cells (Gennari's stripe).

(*c*) The special basilar nervo-protoplasmic plexus beneath the pyramidal cells (sub-pyramidal plexus).

In different regions of the cortex this or that nervo-protoplasmic plexus may be well developed, while the others are less so, but we shall deal with this later on in greater detail; suffice it to say that generally speaking the nervo-protoplasmic plexus in the molecular layer is the one present in *all* cortical regions, and that it is (as we shall show when we come to investigate the brains of vertebrates below mammals), *the oldest and earliest phylogenetically* of the three. We shall further see that with the rise of the brain to the reptilian level the two other plexuses are superadded, these plexuses really overlapping in the reptile brain, but being separated from one another in the brain of the mammal, owing to the vertical growth in depth (thickness) of the cortex. It will be interesting to see that these plexuses are evolved for a definite purpose, and that they have a special physiological significance. Nerve-cells of the cortex, so far as we have already studied them, have besides the *basal nervous axis-cylinder* process, other processes, radiating from the cell body, viz., an apical protoplasmic process, and basilar protoplasmic processes; and when the cells are arranged in strata (as in the cortex), the orientation of the various processes is often very distinct. In some parts of the encephalon cells are found which lack the basilar protoplasmic expansions, but in which the apical expansions are alone present; thus we have Purkinji's cells of the cerebellum, the large nerve cells of the retina (ganglionic layer), the mitral cells of the olfactory bulb, and the cells in the stratum granulosum of the fascia dentata, which all have apical protoplasmic expansion, but lack the basilar branches. Or, again, both systems may be well-developed as in the small pyramidal cells, and the fusiform cells of the polymorphic layer; and indeed, in certain cells of this last stratum, one or more basal processes may develop so markedly as to give the cell a distinct tri-radiate aspect, or the look of an oblique pyramid—approaching the fusiform (*v. ante*, fig. 6). In the ambiguous cell we find also both apical and basal expansions developed; though the former system contains the largest, stoutest, and most expanded branches. We shall here refer to the granule cell: we find that it presents but little development of its protoplasmic expansions at all, these being short, slender, not branched, and frequently looking like mere short bristles or slender spike-like projections from the cell body (fig. 18). As regards the pyramidal cells, we find in simpler and lower types of brains, or in the brains of new-born and young animals, there is but little relative development of the apical system, and less of the basilar expansions. As we ascend higher in the scale of life, from the new-born to the adult, or from the brain of the reptile to that of the mammal, the amount, the extent, and the variety of anatomico-physiological connections of the neurons increase, until, in the human brain, they have reached an extent and a complexity that is well-nigh inconceivable.

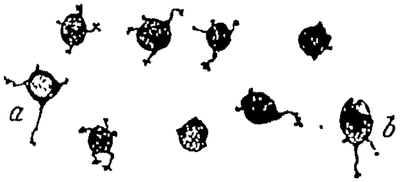


FIG. 18.

Nine granule cells of the human brain cortex stained by modified Golgi's method.

a. is a granule cell in which the section-cutting has removed part of the investing cell-protoplasm, allowing the faintly stained nucleus to be seen.

b. is a cell in which part of the protoplasm is absent, allowing the nucleus to be thus visible at that part.

Primary Sensorial Neurons of Cortex.

We have seen now the main cortical stratum, where incoming sensory excitations affect cortical structures proper in the molecular layer, a region which is everywhere present in the brain, whatever be the topographical cortical variations, and that besides this main nervo-protoplasmic platform, two others exist which all subserve the same purpose, viz., to conduct the incoming nerve excitations to the pyramidal or ambiguous cells of each such sense-recipient area. The important factor to recognise is that two great cortical neuron systems are thus simultaneously aroused into activity, viz., the long pyramidal and the ambiguous cell elements. The sensory nerve currents arriving in the brain as it were bifurcate, they take two main intra-cortical pathways, the ambiguous and the pyramidal. To the study and significance of this we shall return later. We now proceed to deal with the short pyramidal and polymorphic system. The long pyramidal and the ambiguous elements belong to the very basis and foundation of the pallium; they are the oldest phylogenetically, and can be easily recognised in the brains of the *reptile* (lizard) and the *bird* (chicken, &c.), and of course in the brain of every mammal.

Polymorphic System.

The short pyramidal, as well as the fusiform and oblique cells of the polymorphic layer, have all those common characteristics that enable us (in spite of secondary morphological variations in the shape of their cell bodies) to class them in one great group. Owing to the mixed variety of shape as regards cell bodies, this whole layer is well termed the polymorphic layer. We regard it as a new and more recent elaboration in the organisation of the brain, and one associated, therefore, with a special order of cerebral activities. But we must first trace its phylogenetic history, bearing in mind the immense value of this branch of neurology as an aid to all our investigations.

In the brain of the amphibian (frog) but little was known of the histological structure of the cortex; though *Stieda* (*Stud. über das Central-Nervensyst. des Wilbelthiere,* Leipzig, 1870) showed by the carmine method that nerve cells of some sort were present. Later workers, *e.g., Osborn* (*Morphol. Jahrbuch,* 1887), *Belloncini* (1887), and *Edinger (Abhand. de Senckenberg. naturf. Gessell*: Frankfort-on-Maine, 1888, *et. seq.*), mainly studied it macroscopically, or only contented themselves with the tracing of fasciculi to or from it, using Weigert's methods for the purpose as Edinger has previously done for the fishes' brain.

Amphibian Brain.

The first successful attempt to elucidate the cortical structure is due to *Oyarzum,* who, by the application of *Golgi's* method, obtained certain results both as regards the nerve and glia cells in the frog's brain, which he made the subject of a paper in 1890 *(Arch. f. Mikr. Anat.* bd. xxxiv.*)*, and the subject has also been further studied by *Ramon y Cajal* ("Pequanes Contribuciones al conocimiento del sistema nervioso," Barcelona, 1891), also with the aid of Golgi's methods. The following facts are now known. There are three layers of the frog's brain, from pial surface to ventricular cavity, viz., two layers of nerve elements, and one (deep) layer of ventricular epithelium. The nervous layers are: first, molecular layer, and second, layer of nerve cells, which, according to Cajal, are to be looked upon as pyramidal cells, but which the present author ventures to look upon rather as the equivalent of the ambiguous cells (fig. 19). These are cells of an ovoid or globose form, in which the protoplasmic expansions arise as two (or three) main branches from the upper part of the cell body, while the axis-cylinder process comes off usually from one side

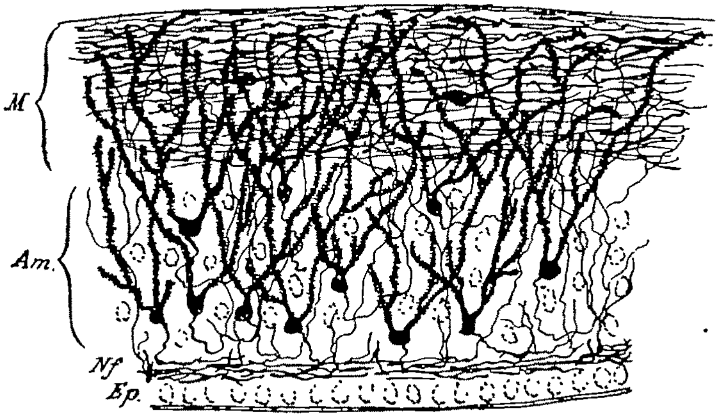


FIG. 19.

Section through cortex of frog's brain (upper part of hemisphere) stained with Golgi's method, and showing the various layers, &c.

M. Molecular layer.

Am. Ambiguous layer (globose-bicornate cells, without basilar protoplasmic processes).

Ep. Epithelial layer (ependyma).

N.f. Thin stratum of nerve fibres.

of the base. The apical expansions branch and ramify and finally end in the molecular layer. There are no basal protoplasmic expansions at all (see fig. 19.) The axis-cylinder process after running laterally some distance, turns up and passing through the layer of ambiguous cells branches out in the molecular layer. These cells are thus all association cells, connecting one point of the cortex with another on the same side, and careful observations reveal the fact (negative) that they give off no commissural fibres, for there is no corpus callosum at all, no transverse connection of the pallium or hemispheres. Further, the only nerve whose fibres arrive and get distributed to the cortex, is the olfactory nerve. We look upon the cerebral envelope of the frog, therefore, as one great central expanse of the olfactory apparatus, as a region for the reception and storage of olfactory excitations, and of response thereto *viâ* the basal olfactory mass the corpus striatum. As we trace the pallium towards its basal attachment, viz., the corpus striatum, we find nerve elements occurring in that region which are different in that they give off long axis-cylinders, which, passing backwards, pass into the posterior parts (post-pallial regions) of the encephalon, and are probably of the nature of *projection fibres*. [The student should here refer back to and read what we have said before concerning the terminations of the olfactory radiations in the mammalian brain.]

Reptilian Brain.

Passing next to the *reptile brain* we may notice that of the *lizard*. Here the general cortical structure is higher in complexity. Instead of two we have three nervous layers, *the three fundamental strata* which are thereafter present in all brains throughout the mammalian series, viz., molecular, ambiguous and pyramidal. The present author not having personally studied the reptile brain with Golgi's method, is indebted to microscopic specimens prepared by others, from which a drawing is reproduced. The reptile brain in cortical structure clearly foreshadows the lamination of the mammalian brain, the main difference being—what we hope to establish in the present thesis—that in the mammalian brain an accessory cortical organisation is added, viz., the mixed pyramidal or polymorphic system, which thenceforward in the mammalian scale increases in richness and complexity of structure up to man.

There are the three fundamental cortical layers in the *reptile* brain, taking that of the lizard as a type. These are, first molecular; second ambiguous; third pyramidal (really long pyramidal) (see fig. 20). The organisation of these will be sufficiently seen in the figure, and we shall only pause here to emphasise the distinction between the ambiguous and the pyramidal elements. It will be seen that the ambiguous cells form a dense and well defined stratum of cells, constituting the second cortical layer. The apical expansion of

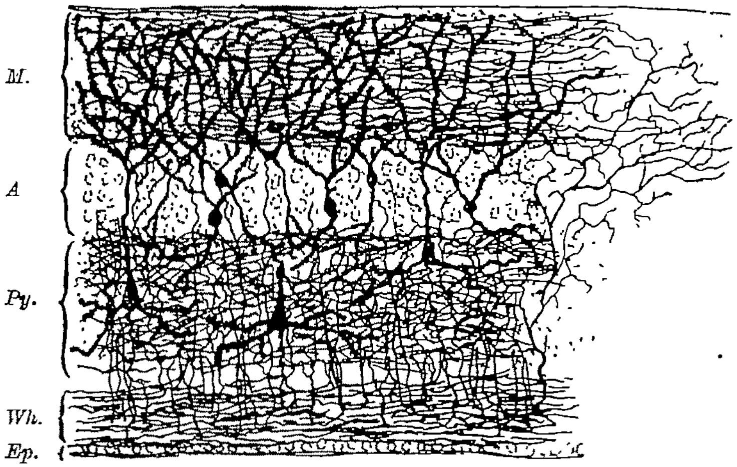


FIG. 20.

Section through cortex of the *lizard's* brain, showing the various layers of nervous elements, &c.

M. Molecular layer.
A. Ambiguous layer (cells about six deep).
Py. Pyramidal layer (long pyramidal system).
Wh. White substance.
Ep. Epithelium (ependyma).

these cells diverge immediately from the cell body as two stout oblique horns (asymmetrical bicornate cell) or from a very short apical stem, in which latter case the cell tends to assume the fusiform shape. Note, also, that while the apical expansions contribute to the formation of the nervo-protoplasmic plexus in the molecular layer, the less developed basal-protoplasmic processes pass downwards into the subjacent region, viz., the pyramidal region. In the form and character of the cell body, the nature and distribution of the apical and basal processes, in their topographical condensation into a stratum of cell bodies immediately underlying the molecular layer, and indeed in every respect, these cells are so characteristic that it should be impossible to fail to recognise their distinctiveness as a special class of cortical neurons, distinct and separate from the next, the pyramidal system.

This, the third layer of the cortex (fig. 20) is composed of the pyramidal system, and here in the reptile brain we find only the pyramidal cell which we have before described as the cell with long apical process, *i.e.*, reaching the molecular layer. All the cells we see here are of this type. What we here notice in the lizard's brain is the beautiful development of these long pyramidal cells forming a conspicuous layer, easy to recognise even with carmine stains, noted, therefore, and well described by various authors, notably Edinger. Each of these cells exhibits a descending axis-cylinder, which passing downwards, enters the white substance (fig. 20). The whole of this pyramidal zone is pervaded by a rich nervo-protoplasmic plexus which, in the reptile, is not yet separated or segregated into two levels, but which, as we have before seen in the mammalian brain, gets segregated into an upper stage (nervo-protoplasmic plexus beneath the ambiguous layer), and a lower stage (sub-pyramidal plexus) (*v. infra* fig. 22). With the higher growth of the cortical system such as we find in the mammalia that is what really happens, the increasing development in extent (vertical depth) of the long pyramidal system, carrying, so to speak, downwards the nervo-protoplasmic plexus of the basal system of the great pyramidal cells, which, therefore, in the human brain (Rolandic area) we recognise as a thick and dense plexus region (of medullated fibres mixed with the basilar protoplasmic expansion of the larger pyramidal cells), as shown by Weigert's and Golgi's methods (fig. 22). A small portion of such nerve fibre plexus (selectively stained with Golgi's method, is shown, fig. 21). Let us add that with this higher development of the reptilian brain (higher, that is, as compared with the amphibian), a corpus callosum also begins to distinctly develop, and with it we have commissural fibres passing across from one hemisphere to another, in addition to the more primitive and onesided association fibres which the amphibian brain possesses. Let us also state that projection fibres are present, passing, certainly, from the pyramidal cells of the cortex, downwards *viâ* the white substance, to distant parts of the encephalon. We have indulged in this, perhaps, rather fastidious exposi-

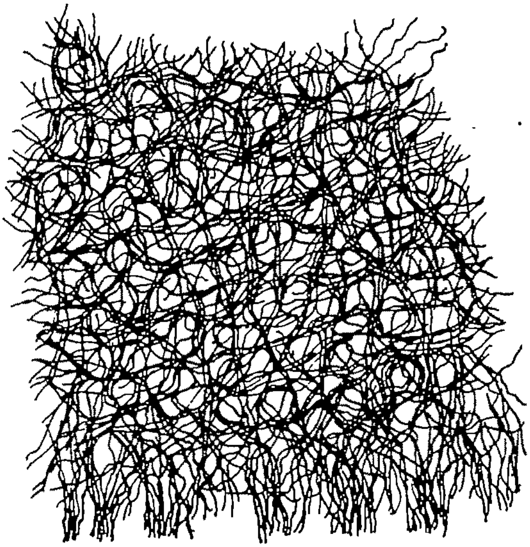


FIG. 21.

Plexus of nerve fibres in the human brain cortex (fifth layer) selectively stained with Golgi's method, to show nerve fibres only.

Note the looser parts of the meshwork: these represent spaces lodging the cell bodies of the nerve cells.

tion, with the object of showing, not only how the structural elaboration of the cortex rises in grades from the amphibian *viâ* the reptilian to the mammalian, but also to draw attention to the significant fact which we earlier tabulated, viz., how, with the commencing quantitative predominance of the cortex (in amphibia) we pass through the larger organisation of the reptile brain before the mammal can be reached, in which the cerebral growth still farther preponderates, reaching its acme in man.

Foreshadowing of the Mammalian Brain.

There is not only a *quantitative* (macroscopic) growth, but a *qualitative* (microscopic) elaboration of the cortical mechanisms as we rise up to the level of the mammalia. Is not this a most striking and significant fact, and one of the deepest import, one which ought to press upon the worker the importance of the comparative method in neurological investigation? We have said, and we have shown proofs for the statement, that the reptilian cortex foreshadows the fundamental anatomico-physiological organisation of the mammalian cortex; and that the three fundamental cortical layers, the molecular, ambiguous, and long pyramidal, are common to both. The further conclusion we are led to from our own investigations is irresistible, that over and above these fundamental cortical strata the mammal possesses another system of cortical neurons which is new, which finds no counterpart in the reptilian brain, and which in the highest mammalian brains attains a wonderful extent and complexity of organisation; we refer to what we have called the mixed pyramidal or polymorphic system, cells with short apical expansions in the sense we have previously explained. It now remains, therefore, for us to study these before we can complete our general survey of the whole cerebral organisation. And here our ground will be widened and cleared by a general survey of some of the features in the organisation of mammalian brains. (*a*) We have first an increase of cortical grey expanse, until in man there is much complexity and depth of convolution of the grey substance. (*b*) An increase in the development of the corpus callosum (inter-hemispheric commissural system) from the somewhat reptiloid monotremata, up to the higher mammalia. (*c*) An increase in the whole *cerebral mass*, both absolutely and relatively to the rest of the encephalon, from the lower mammals up to the higher mammals and man. The white matter of the cerebrum is largely composed of association fibres which interweave in the most complex manner, and connect this or that cortical area with a number it may be, of others. In the human brain these form the main—or at any rate a considerable—bulk of the white fibre systems; the others being the callosal system, and the upward and downward projection system. While many of these association fibres and commissural fibres are derived from the long pyramidal and the ambiguous cells, the whole system of mixed pyramidal or polymorphic cells contributes largely also to the association system.

Meynert's Association System.

Indeed, that the deeper fusiform cells of the last layer of the cortex were thus connected was deduced by Meynert from certain anatomical grounds, to some extent of a speculative nature, but which, as we shall see, have turned out to be in the main correct. Arguing from the fact that the shorter association fibres between neighbouring convolutions (arcuate fibres) pass in curved fashion round the bottoms of sulci to reach the tops of convolutions, he showed that the fusiform cells of the deepest cortical layer are situated amongst these arcuate fibres and correspond with them in direction. Such fibres he terms "fibræ propriæ," and adds, "these bundles of the cortex do not necessarily extend simply from one convolution to the one next adjoining, but they may skip one, two, three, or an entire series of convolutions, and may thus join convolutions which are united among themselves to a convolution lying at some distance from these. The shortest fibræ propriæ lie nearest to the cortex, the longest at the greatest depth, and are separated from the cortex by other intervening fibræ propriæ, the length of which increases gradatim from the surface inwards. The fibræ propriæ or arciform fibres, must therefore be divided into short and long ones, this division answering to a difference in shape, for the shortest fibres alone present the U-shape, which results from their close adaptation to the walls of a convolution depression." ("Psychiatry," 1885, p. 39). Amongst such association fibre-systems Meynert describes the cingulum, the fasciculus arcuatus of Arnold, the uncinate fasciculus, &c.—of which a *resumé* with diagram will be found in Quain's "Anatomy," vol. iii., part 1; "Brain and Spinal Cord," 1893 (pp. 165-166). Similarly as regards the commissural or inter-hemispheric connections, *viâ* the corpus callosum and anterior commissure, the same holds true, that any convolution or cortical area of one side may be connected with its fellow of the opposite side, or (as we now know from researches with the degeneration method) with another convolution (not its fellow) of the opposite side. A cortical area or convolution being thus possessed of an extraordinary range and variety of connections with other cortical areas or regions of the same or opposite hemisphere, rises at once high in the physiological scale according to the extent and number of other centres to which it can transmit nerve excitations.

Greater Extent of Connections.

Meynert supposed that the function of the corpus callosum was to connect only homologous cortical area, *e.g.*, a part of one convolution with its exact fellow of the opposite side only—a supposition which appeared feasible, and one which illustrates the danger of such feasibility—while recent work, especially with the use of *Marchi's* method, has shown that the problem is really a much more complex one, and that the connection, instead of being symmetrical, is really of a most extensive, multiple and wide-spread character, such as Meynert never even thought of. The cell types present in the polymorphic system can all be reduced to one or two main forms, *viz.*, the *pyramidal*, passing by a transitional grade, the oblique pyramid, to the apparently *fusiform* cell. We need not delay in describing the main individual and the transitional forms, for much of it will appear from a study of our drawings before (see fig. 6).

Evolution of Polymorphic Cell Types.

But the present author cannot agree with the contention of Meynert—followed by other authors, *e.g.*, Obersteiner—that the pyramidal type we are considering is to be regarded as a *derivative from the spindle type.* Thus he states, " the form of the pyramid which is most frequently seen is deceptive. The true form is that of a spindle, the axis of which lies in the direction of the radiating projection-fibres of the medullary portion of the convolution " (Stricker's " Histology," vol. ii., p. 387) ; and Meynert further supposes that out of these fusiform cells the pyramidal form is evolved by the development of basi-lateral processes (*vide Meynert, loc. cit.* ; also *Obersteiner,* translated by Hill, p. 353). On the other hand, we find from our own investigations that the order of evolution is somewhat different. Starting from the pyramidal form, *i.e.*, with well-developed apex-process, *and less developed but symmetrical basi-lateral processes,* the cell which is now in shape a young pyramidal cell undergoes an asymmetrical change. One or other of its basilar processes undergoes relatively considerable development, thus initiating an obliquity which many cells continue to exhibit unto adult life (see fig. 6). Such a greatly developing basilar stem will often carry the axis-cylinder with it, the latter thus seeming frequently to originate not strictly from the cell body, but from the thicker proximal part of this basal stem. With this predominance of growth of the one basal stem and the deficient growth of its former congeners, the stage is reached whereby the cell, passing from its original elongated and symmetrical pyramidal form becomes an oblique pyramidal cell, which further gradually attains a pseudo-symmetry which is altogether secondary, by becoming an apparently fusiform cell, such as we see in fig. 5. Repeated observations on the evolution of these forms in new-born and young kittens, have convinced us that the above is the fundamental line of evolution for the morphology of these polymorphic cells, and it is partly with that view, and to emphasise its actual meaning, that we have ventured to term the polymorphic cells elements of this sort, cells of the "mixed pyramidal" type. But a minor number of these cells here and there show minor irregularities of cell-body—departures from the main morphological line of march—some assuming a triradiate, others an inverted pyramidal, and others a more or less irregular contour. These minor variations, however, should not blind us to the real facts of the case, or prevent us from regarding these as belonging to the same "mixed pyramidal" system.

What are the topographical limits of this system of cells? and what their relative development in different cortical areas.

While these cells form a well-defined stratum—often of considerable thickness — in the deeper moiety of the cortex, their upper and lower limits are not very precise.

Upper limit of the polymorphic or mixed pyramidal cells.—The cells on the upper confines of the polymorphic layer do not form a well-bordered line; they interpenetrate somewhat into the territory of the superjacent great pyramidal cells; but the extent of such invasion or interpenetration is never great—seldom more than the lower fourth of the long pyramidal layer. When we pass higher up into the midst (middle region) of the *long pyramidal system,* hardly a cell of the polymorphic system can be seen, and when we approach the lower confines of the ambiguous layer not one such can be seen. The mixed pyramidal cells stop short long before, and the pyramidal cells which we do see—small pyramidal cells—in the confines of and intermixed with the lower ambiguous cells, are quite another in character: they are the upper cells of the long pyramidal system. Repeated examinations of a very large number of preparations from several human brains, and from the brains of various mammals (cat, rat, rabbit, ox), have shown us that this is almost universally true. The mixed pyramidal or polymorphic cells keep to their own segregated cortical territory, constituting the lower moiety of the cerebral cortex.

Lower limits of the polymorphic system. The lower cortical limits of these cells is exceedingly hard to define—nay, almost impossible, especially in the cortex which forms the top of a convolution. As we approach the white substance these cells get sparse and few in number, but never end in a distinct stratum. Here and there a few such cells, straggling as it were—subcortical in position—can be recognised, and isolated ones of the same sort scattered in all the depths of the medullary substance of the convolution, several millimetres away from the lower gray limit of the cortex.

Migration of sub-cortical Cells.

We are inclined to think these cells are possessed of migratory power even in new born and young animals, and that they can pass upwards from more sub-cortical regions into the deeper cortical stratum. If a fœtal kitten about a week before full term is removed, and its brain prepared and examined by the Iolindine method of staining, we recognise these cells in long straggling array at all distances from the cortex, and fairly abundantly scattered. Indeed, even in the midst of the cerebrum ovale, in the very centre and core of its texture, such cells can be recognised. A week later, in the brain of the new-born kitten, they condense more and more into the deeper moiety of the cortex ; the sub-cortical straggling cells lagging behind are fewer in number ; and in the kitten a few days or weeks old they are yet fewer and more sparse. In this respect our observations certainly force on us very strongly the conviction that these cells behave in the above manner, forming as it were a sub-cortical growing system, later, not only in phylogenetic history—as we have shown before—but still growing (not numerically) in elaboration during even the early days of extra-uterine life. We cannot at present give any positive conclusions concerning the significance of this phenomenon, but the question naturally arises in our mind—

"Are these the cells which belong to the fundamental association systems of the brain—as Meynert thought—or (as we feel inclined to think) are they a new and more recent portion of the system—*i.e.*, an accessory association system?" We have seen from our studies on the amphibian and reptilian brain that the older system of cortical elements—the ambiguous cells and the long pyramidal cells—are competent to, and do of themselves give rise, not only to association fibres between different points in the hemisphere (inter-hemispheric), but to the callosal or commissural system of fibres (inter-hemispheric fibres). They are not mere or exclusive projection fibres as Meynert thought they were. They give rise to the oldest association and commissural system in the brain of animals, and it is only when we rise to the scale of mammals and man that we find an accessory system of nerve elements superadded—or shall we say subadded—to the cortical structure—viz., the mixed pyramidal system of cells, and with these their association fibres—in other words an accessory association system. These therefore belong to the cortical *substructure*, the latest evolution in the cortical mechanism, though the lowest of the cortical strata in topographical disposition.

Comparative Growth of the Polymorphic System.

And, we may add, the mixed pyramidal system of cells and their connection increase in relative growth and elaboration as we ascend the mammalian scale, finding their highest expression in the human brain.

We can arrive at a rough approximation of the truth of this by noting the following series of mammalian brains in an ascending scale, viz., *rabbit, cat, monkey, man;* and taking any one area for comparison, *e.g.*, the pre-central region in the last three, to which the extra-limbic area of Lewis in the rabbit's brain corresponds. We shall find the thickness of the polymorphic layer is approximately, rabbit $=\frac{1}{6}$, cat $=\frac{1}{5}$, monkey $=\frac{1}{4}$, and man $=\frac{1}{3}$ to $\frac{1}{2}$, relative to the whole thickness of the cortex. The precise evaluation of the thickness of this mixed pyramidal stratum is often a matter of difficulty owing to the fact that its lower border is not so sharply demarcated as we would wish; the above estimate must therefore be taken only as approximate. Yet there is no fact more striking than the one that as we pass up the mammalian series from the level of the rodent, *viâ* the carnivora to the primate, including man, the textural thickness and complexity of the polymorphic layer rises, relatively to the rest of the cortical organisation, until in man it attains its highest relative and absolute evolution. The author is of opinion that future research in this direction will further tend to show the importance of this item of the cerebral organisation in man, not only in general neurology, but in neuro-pathology, and the studies of the *various insanities* and *system diseases of the brain.*

Fields of Conjunction.

The whole depth of the polymorphic stratum is pervaded by a plexus of nerve-fibres of striking richness and extent, the fibrils and terminals of these fibres interweaving throughout with the apical and basal protoplasmic expansions which each polymorphic cell gives off. This nervo-protoplasmic plexus is generally of a diffuse nature, but it exhibits some condensation in two regions—(*a*) the sub-pyramidal region, or rather in the boundary zone between the pyramidal and polymorphic systems. This is the region common to the lowest (largest) pyramidal cells and the upper polymorphic, for as we have stated before the upper polymorphic cells inter-penetrate a little way in between the largest pyramidal cells. The cell bodies and basilar expansions of the largest pyramidal cells, and the cell bodies and processes of the upper polymorphic elements, lie in this plexus region—the thickest and densest in medullated fibres of all the cortical plexuses. This is remarkably shown not only by Weigert's method, but also with Golgi's silver method, and we have already (fig. 21) given a drawing of the appearance of a portion of this plexus of nerve fibres as seen with Golgi's method.

The following figure, constructed after a careful and detailed study of the cortical nerve fibre systems, both from Weigert and Golgi specimens, will give an approximate idea of the relative complexity of this region (fig. 22). Careful observation further shows that though the nervo-protoplasmic plexus *beneath* this condensation is diffuse, yet in the very *heart* of the polymorphic region—far away therefore from all long pyramidal cells, a second and somewhat slight condensation takes place. The level of this is approximately between the *great* nervo-protoplasmic plexus (subpyramidal) and the white matter (fig. 22).

The whole cortex is thus pervaded by several strata of these nervo-protoplasmic plexuses, extending sheet-like throughout all the cortical grey, each of definite anatomical structure, and each playing the part of a "field of association or conjunction" wherein the collaterals and terminals of *nerve fibres* (association, commissural, or projective) comes into relationship with the apical or basilar protoplasmic expansions of cortical nerve cells.

Loss of Insulation in such Fields.

We have one fact to emphasise in this connection, and that is the fact that in these fields of conjunction the medullated nerve fibres end not as such, but as *naked* collaterals and terminal fibrils; the myelin sheath stops short, it does not invest these fine collaterals and terminals, which thus are allowed to come into the most intimate contact with the (also) naked protoplasmic expansions of the nerve cells. The fibre is insulated throughout its course, *i.e.*, covered by myalin; but in the terminal region, *i.e.*, the field of conjunction, the insulation ceases, and by the intimate contact of the nerve fibrils and collaterals with the expansions of the cell protoplasm, nervous excitations propagated along these fibres are capable of exciting the cell.

New Activities in such Fields.

This excitation of the cell is, however, to be looked upon as a new phenomenon. For *first*, it (*i.e.*, the cortical cell) is

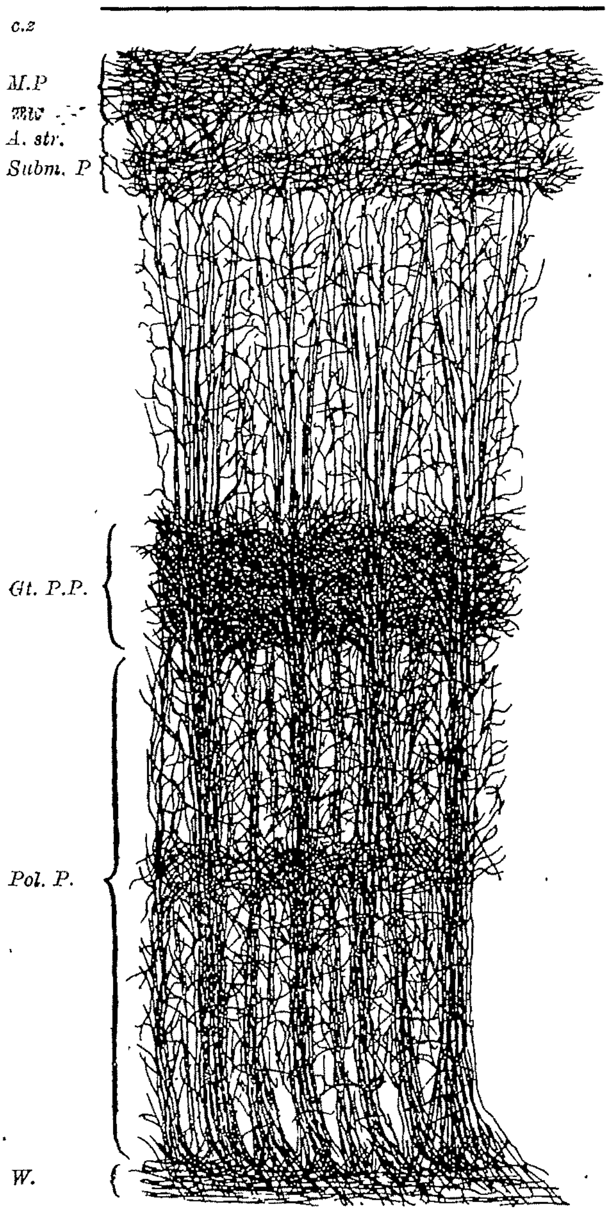


FIG. 22.

Cortex of human brain showing the nerve fibre systems and plexuses (combined Weigert's and Golgi's methods).

c.z. Clear zone (free of nerve fibre).
M.P. Molecular plexus (Exner's), in the molecular layer
A str. Ambiguous cell stratum. *Subm P.* Sub-molecular plexus.
Gt. P P. Great pyramidal plexus. *Pol. P.* Polymorphic plexus.
W. White substance.

a distinct element anatomically. *Second*, the activities of cortical cells are accompanied by psychical phenomena of various kinds. *Third*, the cortical cells and centres have —at least some of them—a rhythm of their own. *Fourth*, the cell activity is accompanied by considerable physicochemical changes, greater in magnitude than those accompanying the mere passage of the nerve currents along nerve fibres. The nerve fibre during the passage of nerve currents along it does not rise appreciably in temperature; the chemical products formed during its activity are exceedingly small in quantity. On the other hand, the nerve cell aggregates or nerve centres during their activity produce much heat and give off many chemical metabolic products (see Schäfer's Address at the Neurological Society, January, 1893, "On the Nerve Cell as the basis of Neurology"). Hence, mass for mass, the cortex is more abundantly supplied with blood than the white substance of the brain. The nerve cell (cell body and protoplasmic branches) is an apparatus where energy is elaborated and stored as it were under high pressure, and from which discharges of energy (nervous discharges) may take place in two or three ways, viz., (*a*) as *the result of direct excitation* by incoming nerve currents (presentative stimuli); (*b*) as *the result of its own (rhythmic) nutritive activity*, apart from the influence of incoming excitations, that is, its own intrinsic nutritive rhythm, the result of nutritive accumulations of energy, and which is a fundamental property of protoplasm whether nervous, muscular, or epithelial; (*c*) as *the result of the revival of previous nerve activity*, caused at that time by presentative stimuli, repeated or organised—stamped, as it were, into the nerve elements—until *now* the element so altered is able to give rise to nervous discharges *in the absence* of such presentative stimulus, and as the result of its own present nutritive activity. The study of nerve centres and their activities, even in such low forms as the *medusa*, and in higher forms (fishes, amphibia, mammals) shows that these three fundamental forms of nerve activity underlie the whole of the animal kingdom. (The reader should, for an excellent study and exposition of this, read Horsley's Fullerian Lectures for

1891, "On the Functions of the Spinal Cord and Bulb.") Their special application in the study of brain action (psychical function) we shall later refer to when dealing with the law of psychogenesis. Our survey of the anatomico-physiological constitution of the cortex and sub-cortical connections are now sufficiently advanced to enable us to follow the pathway of a sensory excitation, visual, olfactory, musculo-cutaneous or kinæsthetic, arriving at the cortex *viâ* one or other of the upward projection systems (optic, olfactory, or fillet radiations).

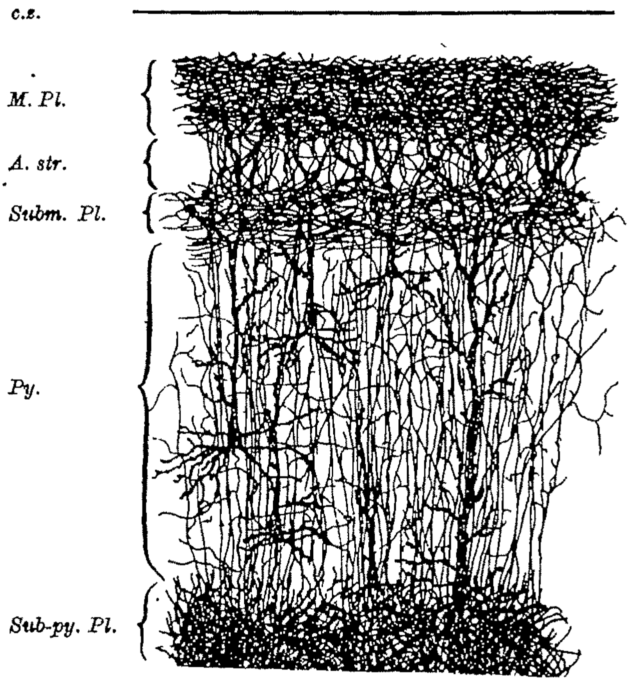


FIG. 23.

Showing the ambiguous and long pyramidal cells, and the nervo-protoplasmic plexuses belonging to them.

c.s. Clear zone.
M. Pl. Molecular nervo-protoplasmic plexus.
A. str. Stratum of ambiguous cell (3 are shown).
Subm. Pl. Sub-molecular plexus.
Py. Deep stratum of long pyramidal cells (four are shown and in its deeper part two "short pyramidals cells" are also shown).
Sub-py. Pl. Sub-pyramidal plexus (of which the upper *half* is only shown).

Sensory Sequelæ.

Such nervous excitation (say a visual one) arrives at its proper cortical "sensory centre," and, as we have seen, spreads out in the molecular and sub-molecular layers. Here it affects, therefore, intrinsic cortical elements, and in the following way. In the molecular layer (see fig. 23), the incoming sensory terminals come into relationship with the whole apical expansions of the ambiguous cells, as well as the peripheral tufts of the long pyramidal cells. In the sub-molecular layer the whole of the basal expansions of the ambiguous cells, and a portion of the apical expansion of the long pyramidal cells, are affected. These cortical elements, therefore, viz., the ambiguous and the long pyramidal cells, are thereby excited to discharge. The *excitation, rise of nerve-tension, and discharge* of the conjoint ambiguous-pyramidal elements thus affected is the nervous process, the psychical counterpart of which is a *sensation* (in this case a visual sensation) aroused in the mind.

Specific Sensations.

Such a sensation may be *faint* or *vivid*, according to the intensity of the cortical *excitation and discharge*. Similiarly, according to its cortical *locality* the sensation may be a *visual*, a *muscular or cutaneous*, an *olfactory*, &c., or other sensation. That such *an excitation followed by a discharge* (nervous) of a primary sensory area will be accompanied by its appropriate *sensation* (psychical) is now a well-established fact based on experimental and pathological grounds. The experimental data are available in text-books of physiology. We shall here briefly refer to the work of neuro-pathologists in this connection, beginning with the immortal work of Hughlings Jackson on "Local Cortical Discharges (Jacksonian epilepsies)."

Jacksonian Epilepsies.

It was thirty years ago that *Hughlings Jackson* taught (1864) that certain forms of epilepsy with local motor disturbances were almost uniformly due to organic disease of some kind, situated in the Rolandic region of the brain; a view comparatively neglected then, but which the startling revelations of the experimental school of workers (Fritsch and Hitzig in 1870, Ferrier, 1872, and '79, and others) have since served to call attention to. And following these a brilliant school of clinico-pathological investigators such as *Charcot* (Charcot et Pitres: "Localisation cerebrales," *Revue de Medicine*, 1879 and 1883); *Nothnagle* (Topische Diagnostik der Gebirn Krankheiten, 1879); *Wernicke* (Gehirn Krankheiten, 1881; *Ferrier* (Localisation of Brain Disease, 1878); *Roland* (De l'Epilepsie Jacksonienne, 1887); *Allen Starr* ("Cortical Lesions of Brain," *American Journal of Mental Science*, 1884); and others, have by their researches conclusively proved the wonderful truth and correctness of the Jacksonian doctrine while adding important confirmatory and other details. But the great and important feature which we have to recognise is that from the vast accumulation of such details, not only have the various centres which regulate muscular movements (kinæsthetic areas) of the organism been thus more accurately localised in the human brain, but also the other important centres which have to do with the reception or perception of gustatory, olfactory, visual, auditory, and cutaneous sensory excitations, have each and all of them received its proper and appropriate "local habitation." And thus the field of the Jacksonian epilepsies has been considerably extended, and now includes those not only starting from this or that focus of the kinæsthetic areas, but also each of the many other foci which are the cortical centres in relation with the other senses mentioned above. And thus it happens that just as when a kinæsthetic focus is aroused the psychical counterprocess that is evoked is a sensation (either a cutaneous sensation or a sensation of movement), referred to this or that peripheral body organ (face, hand, foot, &c.), which is in relation with the said focus, so if the focal discharge be stronger the nervous excitation is propagated along the pyramidal tract, and arousing the motor bulbo-spinal centres, produces one or other movement of the speech organs, face, arm, hand, or foot, according as to whether the seat of the cortical discharge is the tongue, face, hand, or leg area.[1] And similarly with a slight focal discharge occurring in this or that special sense cortical area, we have aroused its psychical accompaniment, viz., a hallucination of this or that sense—gustatory, visual, olfactory, auditory, &c. To particularise, we may add that when in cases of such epilepsies the primary disturbance is a hallucination of hearing, the cortical seat of disturbance is in the posterior part of the two upper temporal convolutions, when a luminous or other visual excitation, the cortex involved belongs to the calcarine or other part of the occipital region, when attended with the seeing of words or letters the disturbance is in the angular region (visual word centre), and when attended with smells or taste the cortical region of disturbance is in the inferior and distal portion of the temporo-sphenoid lobe. We have thus kinæsthetic epilepsies ; we have also special sense epilepsies, and to complete the picture we may add—we have psychical epilepsies. Not that the other epilepsies are non-psychical, for each and every one of them is attended by its own psychical equivalent, viz., a feeling of cutaneous excitation or of a peripheral movement or the excitation of one or other special sense.

Psychical Epilepsy.

But in psychical epilepsy in the stricter sense of the word, there is a disturbance of a more distinctively psychical nature, consisting either of a moderate mental disturbance (bewilderment, sudden loss of memory, stupidity, and a dreamy state, or a state of reminiscence—the intellectural aura of Hughlings Jackson), or of a more serious and violent maniacal excitement or intense furor in which the patient may do the wildest and most awful deed, homicide or even suicide, as the outcome of the intense cerebral (psychical) discharge affecting the higher regions of his brain.

---

[1] The number of cases operated on for Jacksonian epilepsy are now (1885-94) over 800 (about 400) as known to medical literature. (See the writings of MacEwen, Horsley, Bergman, Lucas Championiere, and Allen Starr.)

We cannot exclude this form, psychical epilepsy, from the category of the other special sense epilepsies: Our aim and object should rather be—following the light we now have—to work out its localisation in the brain, to ascertain what area, or what set of cortical elements is the seat of such epileptic discharge.

Sensory Processes in the Rolandic Area.

Of late years the more accurate clinical study of cases with destructive lesions of the Rolandic cortex, and the stimulation (in man) with the faradic current of the Rolandic cortex without anæsthetic (Dana, Ransome), have shown that this cortical region is, at any rate, partly a sensory area for the reception (*viâ* the fillet radiations) of incoming sensory currents from the face, arm, leg and other movement organs. In *Ransome's* case (Congress of Experimental Psychology Proceedings: London, 1892, pp. 89—96, paper and discussion), the chief results obtained were:

(1) *Contraction* of groups of muscles in hand and arm by a moderate current;

(2) The production of a *sensation* in those parts where a weaker current was used, contraction being added as the current was strengthened;

(3) Improvement in the muscular sense during and after stimulation;

(4) Weakening of voluntary motor power after a strong induced contraction.

Dana has also recorded cases (*Medical Record,* May, 1893), of such faradisation of the Rolandic cortex, with similar results. Fortunately, in the Rolandic area we can trace the further result of the cortical discharge and its anatomical pathway. The excitation and rousing into activity of its superficial cell elements by mild faradisation, viz.: those entering into the constitution of the molecular and sub-molecular plexuses, is accompanied by a cutaneous sensation like tingling and formication, referred to a peripheral organ.

With a stronger stimulus to ensure a discharge from the long pyramidal cells (downwards, *viâ* the pyramidal tract), the result is a spinal cord excitation issuing in a peripheral movement. The circle of phenomena is then completed. The movements thus effectuated in the peripheral limb give rise in their turn to a returning cycle of impressions arising from the limb muscles, and arriving *viâ* the sensory roots and fillet to the Rolandic area. *After* such a movement is effectuated does the subject become conscious of its occurrence: that is by the returning cycle of impressions arriving to the brain from the limb muscles? These incoming messages which inform the brain of the movements of the limb, arrive (strictly speaking) not in the pre-Rolandic but in the post-Rolandic (ascending parietal) convolution (see Hosel's paper in *Neurologische Centralblatt*, July, 1894). In the pre-Rolandic or ascending frontal convolution, and in the adjoining posterior portions of the three frontal convolutions, as well as the prolongation of these areas on the mesial (marginal) convolution, we find the last term in the cortical series, the finally disposed executive mechanisms—the δουλος, as it were, of the other and higher cortical areas, and the common servant of them all.

Results of Visual Excitations.

First, the visual excitations arriving in the calcarine region and arousing into activity the cortical elements, then exciting these to discharge, the psychical accompaniment of which is a primary visual sensation. From the primary (calcarine) visual area, the discharge spreads to associated visual areas. These are spread over and occupy a considerable surface of the *general occipital* and *angular* gyri in the monkey and in man. Some of the anatomical tracts connecting the primary visual area with other areas of the same side have been actually demonstrated. Thus *Vialet* has shown that from the inferior calcarine (or lingual) region a distinct fasciculus of fibres passing transversely outwards from that region, beneath the occipital cornu of the ventricle, is distributed to the second and third occipital convolutions of the external surface (*Société de Biologie*, Paris, July, 1893 — quoted in *Revue Neurologique*, May, 1894); and Beevor has shown in the marmoset (an American monkey) another fasciculus from the calcarine region which passes *forwards* (not to be confounded with the optic radiations), into the centrum semi-ovale, and might connect this part of the cortex with other parts of the visual centre (? the supra-marginal and angular gyri) (*Phil. Trans. of the Royal Society*, 1891, B. vol. 182, p. 135). Further, the experiments of Schäfer and Barrett (*Journal of Physiology*, 1889), have shown that a connection exists between the excitable areas on the external occipital convolutions, and certain conjugate head and eye movement areas of the upper and middle frontal convolutions, and by an ingenious set of experiments they have shown that at least some excitations of the occipital area pass to the frontal area and thence evoke the ocular movements in question. All these put together enable us to trace in a measure the further pathways of the visual excitation after it has evoked the activity of the primary visual (calcarine) area. For the nervous excitations from thence may pass outwards into the external occipital convolutions, and rouse their activity; or, forwards into the angular region, rousing to activity the nervous elements there, elements which we know especially in man to be associated with the psychical function of *word-seeing and understanding*. On this latter function the clinical and pathological evidence to hand is now so complete as to form an interesting chapter in the subject of aphasia—a study which must be looked upon as a pathway to the understanding of the pathological mechanism of some of the insanities.

The areas of cortical elaboration associated with the visual sensation are thus in man of great extent, covering large and extensive areas of the calcarine, mesial and external occipital and posterior angular regions. Similarly the auditory distributions cover the posterior half of the two upper temporo-sphenoidal convolutions. And accordingly as one or other cortical centre is excited we have as its psychical equivalent a sensation (*v.* supra in *re* Jacksonian epilepsies).

Aphasia.

And similarly if the connecting tract between any two centres is destroyed, excitations aroused in the one centre cannot pass into the associated centre, and therefore the sequence of sensation, or of movement following sensation is lost. In this way—and limiting ourselves to the subject of aphasia alone—various clinico-pathological types are possible, each with a psychology of its own. Aphasias may occur not only from lesions in this or that sensory receptive word-seeing or word-hearing area, but also from lesions involving the *tracts* connecting each of these with its appropriate executive (pre-Rolandic) centre, and we may also get aphasias (and agraphias) from lesions limited to these last alone. (See especially Allen Starr. BRAIN, July, 1889).

Clinical investigation abundantly shows that each and all of these cortical and tract lesions has its appropriate psychological symptom-complexes. The whole body of evidence that we have thus far been investigating leads us to the conclusion that the cerebral cortex is the seat of various psychological activities, different in kind (*e.g.*, visual, from auditory or tactile, &c.), and different also in the form and extent of their elaboration (lower and higher visual and auditory, &c.).

Anatomico-Physiological Evolution of Rolandic Cortex.

Similarly, on the executive side of the brain (Rolandic region) we find, starting from the organisation of this area in the new-born and the young, that as growth of the organism, and perfection of its movements (adjustments to the environment) takes place, a remarkable development—differentiation and integration—takes place in these cortical-centres, which is recognisable and can be demonstrated by microscopic investigation, and which will serve as a type for what is a widespread process underlying the growth and elaboration of the whole cortical mechanism.

In the new-born and young (kitten's brain, rabbit, and other animals) and while the individual cells of the various cortical layers have already reached the completion of their *numerical* growth, the individual *neuron* has by no means reached the acme of its evolution. Growth takes place in two directions (*a*) in size of the cell and its processes; (*b*) in amount and complexity of its anatomico-physiological connections. This latter is a fact of the deepest psychological import, for herein we have a key to the better understanding of some of the profoundest mysteries of psycho-physiological life, for we can see the evolutionary grades in the nervous mechanism which subserves it.

Quantitative Evolution.

Growth in size of the neuron or brain cell. In the kitten, from birth up to a little way into adult life, growth in size of the *cell body* takes place. But this soon ceases; and in the young cat of a few months old, and in the adult one of two or three years the cell bodies do not differ in size. The present writer, from a study of specimens from a young cat's brain (about 6½ months old) compared with those of a full-grown cat rather over one year old could make out no appreciable difference in the following two regions: (*a*) the anterior limb of the sigmoid gyrus; and (*b*) the uppermost arcuate convolution.

Having compared also the cell bodies from the ascending frontal convolution of a child two and a-half years old—a hospital *post-mortem*—and a number of brains from adults of various ages (25 to 50), the present author fails to find any notable difference in the size of the cell bodies. On the other hand, in a fœtus of five months the cell bodies of the cortex are distinctly smaller (*Vignal,* "Developpment des Elements du Systeme Nerveux," 1889), and more closely packed together in the cortex; and what would seem to occur about full term, and in new-born and young animals is as follows:—The cell bodies, up to a certain age, seem to grow in size, but that limit is soon reached while the animal is still young. But meanwhile *the cell bodies* certainly seem to get further apart during early life—much more so than their slight increase of size would be adequate to account for, and which therefore has been explained by various authors as due to increase of *blood vessels*, of *neuroglic*, or of *ground-substance* (Boll).

Qualitative Evolution.

Our own opinion, however, based on the study of a complete series of Golgi specimens in kittens, from the fœtus about a week before term to the young kitten a month old, shows that this phenomenon is correlated not with the growth of non-nervous structures pushing the nerve cells further apart, but *really* with the next factor. We shall now discuss the growth in the extent and complexity

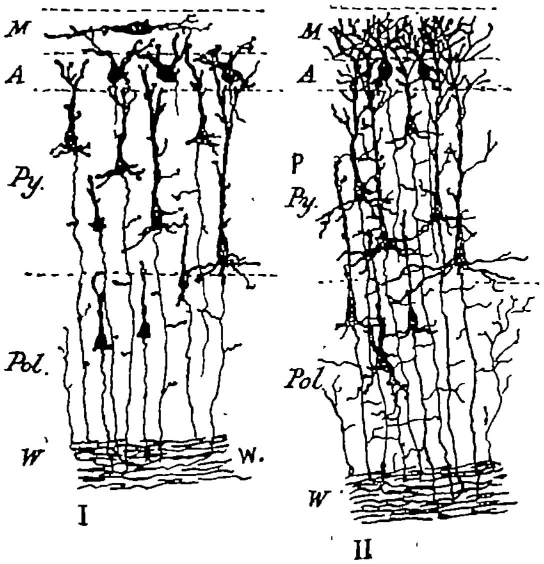


Fig. 24.

Sections through brain of kitten to show the growth in the complexities of the cortical organisation.

I. From brain of kitten about one week before full term.
II. From brain of kitten one day old (after birth).
M. Molecular layer.
A. Ambiguous cell layer.
Py. Pyramidal cell layer.
Pol. Polymorphic layer.
W. White substance.

of the process, protoplasmic and nervous, which belong to the nerve cell, and the full and precise recognition of which is only possible with the use of Golgi's methods.

Growth in the extent and complexity of connections of the protoplasmic and nervous processes of the brain cell. This is the real and fundamental fact, the importance of which is shown by the study of specimens prepared with *Golgi's method.* The growth which takes place during the close (last month) of intra-uterine life, and during the period of infancy, is as far as the cortical nerve elements are concerned *not a numerical* increase of elements ; it is to a slight extent *a quantitative* growth ; but to a main and notable extent it is a *qualitative* growth, at least during the last month of intra-uterine life, and the first of extra-uterine. The nerve cell bodies, which in intra-uterine life seemed close packed and touching one another, get separated and distant from one another, being pushed apart by the extraordinary growth of protoplasmic processes emanating and growing out from the whole of the cell body, both basilar and apical in all directions, and simultaneous with this a parallel growth of collaterals in increasing number and extent from the already formed axis-cylinders of the same cells. The *cell bodies* remaining are numerically the same, the increased growth of their processes pushes them further apart, hence they seem apparently to get more sparse in the growing brain, but this is only a feature which should be interpreted in terms of what we have explained. These complexities of growth will be understood by comparing the pyramidal cells of medium size, taken from the same cortical region, but at different ages of the animal (kitten) : thus (1) at about a week before full term, and (2) from a kitten one day after birth (fig. 24). These drawings, carefully prepared from several sections, illustrate faithfully the main observable differences which are really considerable. In the first, the protoplasmic expansions of the apical system of the long pyramidal cells are still not fully developed, and have hardly reached the molecular layer, and the three ambiguous cells present show expansions which are yet *short, gross,* and ill-differentiated, while the cell bodies are notably plump. The basilar processes from these cells is hardly recognised; they are just sprouting out, and the axis-cylinder process is short and very slender, with no collaterals on it.

The pyramidal cells have basilar expansions which are as yet feebly developed, and an axis-cylinder each, with two or at most three, very short and rudimentary collaterals coming off a short way. In the molecular layer a fusiform cell (Cajal's cell) is seen—with difficulty recognisable at this stage. In a kitten but one day old the growing structural complexity is already becoming manifest (fig. 24, II.). The cell bodies of the various elements have hardly increased in size, but the development of cell processes is conspicuous. The apical expansions of the protoplasmic cells have reached into, and are richly pervading the molecular zone, and the lateral branchlets of the apical stem are also more numerous.

The same holds with regard to the ambiguous cells also. In these latter the basilar protoplasmic processes are also better developed : in the pyramidal cells this is still more notable. All the axis-cylinders show more collaterals, four to five each, and these are rather longer also in their course. The three or four cells of the polymorphic system are also more advanced in structural grade in the latter figure.

In a kitten a month old these changes are still more advanced, and the mechanism more elaborated. My observation in these respects on the kitten's brain are entirely confirmatory of the results obtained by Cajal (1890) from the brain of fœtal and young mice of various ages. They illustrate the important fact that growth in anatomico-physiological complexity is the characteristic of the neuron in early extra-uterine life, a growth, or rather elaboration, which, as further observation shows, is carried on till apparently the adult condition of the animal is reached.

Physiological Elaboration.

By this growth and elaboration of the cortical neurons, certain possibilities are realised. The cortical executive mechanism—as shown in the structural organisation of the Rolandic or sigmoid area—is rendered more perfect for the performance of movements. With the growth of the protoplasmic expansions these latter come into functional association with a greater and greater number of nerve fibrils which pervade the molecular area, as well as—more diffusely.—the various other cortical strata also. They therefore can receive and react to a larger number of incoming excitations reaching these areas from associated sensory areas, or from the upward sensory projection (fillet system) of fibres. And with the increased growth in number and extent of collaterals, the superficial pyramidal cell, like the ambiguous cell, is able to transmit currents (excitations of its own) to the neighbouring and deeper lying pyramidal cells, whose apical or basilar expansions these horizontally running collaterals touch. This serves not only to render the whole body of nervous excitations discharged from the cells down the pyramidal tract more solid, and as it were *en masse*, but by such means of nervo-protoplasmic contact whereby the upper cells can influence the lower in series, capacities for movement are furnished which by the special growth of this or that protoplasmic branch, and this or that collateral and terminal related thereto, can grow into definite and well-integrated nervous mechanisms for the performance of manifold, distinct, and well-differentiated movements such as we know (from experimental work), the Rolandic area is capable of performing.

Education.

With a process partly of imitation and education of the young child or animal, and partly of repeating some movements and series of movements, and not repeating (*i.e.*, eliminating), other movements and series of movements, there undergoes a parallel structural differentiation in the Rolandic cortex (and for the same reason of the other—visual, olfactory, auditory, &c.—cortical areas also). The growth in complexity of organisation and in extent of nervo-protoplasmic contact between the various pyramidal elements of the Rolandic area, we look upon as the correlate of the growth in the elaborateness of movements which the growing organism is learning to effect; its early, weak, and unsteady movements of dyskinesis gradually growing by a process of education and repetition of certain movements, and the non-repetition of others, into the later, stronger, and steadier movements of eukinesis which appear as growth goes on. There is thus a progress in the qualitative growth of the cortical organisation for movement, from the fœtus up to an early period of life (adult life), the precise extent and limit of which we at present cannot delimitate, but of the progress and elaboration of which we are assured.

Extending this doctrine to the other cortical centres, of special sense,—and of the other associated or secondary centres built as it were around the platform of each of these, and further inter-communicating each with many (or all) of its neighbours by association tracts, we can come to faintly realise the exceeding potentialities and capacities for the various forms of sensorial, psychical and psycho-motor life which the active brain is capable of, during a healthy and well-trained life.

Quality and not Quantity.

Thus with the proved anatomical basis for quality of brain, as we have above shown, and as is supported by phylogenetic considerations which we also previously dealt with, a brain small in size (like that of Gambetta), may from its intrinsic high elaboration be able to sub-serve more varied, extensive, and multiform activities in life and thought than others of grosser organisation, even if blessed with more quantity, can be capable of. It must be the work of future investigation to correlate the brain in idiocy and imbecility with the capacities during life which their subjects exhibited, and on the anatomico-physiological lines we have above spoken of. The protoplasmic processes and their granulation, and growth in extent and complexity of connections, and the correlative growth of nerve-terminals and collaterals in the brain cortex are thus of the profoundest significance; the formation of each protoplasmic granule, and the coming into contact of each granule with a nerve fibril, indicating the mile stones—or perhaps the inches and footsteps—which mark the slow advance of cerebral (mental) organisation.

The progress of our investigation is now ripe for our taking up the next great theme, the law of *psychogenesis.*

Law of Psychogenesis.

In the child, the whole progress of growth and education is a long, a tedious, and often a painful toiling and passage from dyskinesis to eukinesis for movements, and similarly from dysæsthesis to euæsthesis for sensation and feeling. As on the executive or psycho-motor side the mechanisms for simple movements grow with the growth of the Rolandic sphere into higher and more elaborated mechanisms which subserve the higher executive phenomena which we term *acts;* so on the sensorial side there is a somewhat parallel development taking place whereby distinctness and vividity of sensations, their sharp integration and differentiation from one another, and the capacities in the organism for their quasi-spontaneous revival (recollection) in the brain are rendered possible. Between these two great spheres the sensorial on the one side, and the executive on the other lies what, for want of a better title we may call a transition region of varying extent, but of the existence of which we are sure from anatomico-physiological, psychological, and pathological grounds ; and we propose to deal with its working in a brief study of the laws of psychogenesis.

The law of psychogenesis :—In the child the march of evolution in the psycho-motor or executive sphere is from dyskinesis to eukinesis, and in the sensorial sphere from dysæsthesis to euæsthesis. These are the functional unfoldings which accompany the structural elaborations which are taking place in the growing brain. In the early functional stages the various sensory currents arriving in the brain excite neurons which are themselves rapidly growing and changing, and which from that fact alone are the seat of a continuous psychical turmoil. As growth and elaboration proceeds, the child's capacities for sense-perception get improved, better defined (*i.e.*, better differentiated from others), and intensified (integrated). It passes through the lower grade of *imperception* (Hughlings Jackson) to the higher grade of *apperception* (Wundt). Parallel with this growth in the receptive (sensorial) sphere is the growth in the executive (psycho-motor) sphere—such growth and elaboration being, in the strictest sense, anatomico-physiological. The psychological state which accompanies this is one in which the empirical ego is not yet distinctly recognised, but is (by the child) referred to in the third person. This is the characteristic of the preconceptual stage: it is the state in early childhood. On the brain this formed and forming, the external logic of events has a remarkable influence. The child comes more and more into contact with its environment, and the various objects in the environment tend to be presented again and again—individually. But a further stage now occurs when two or more objects in the environment happen to occur together. The apposition of two events without, *i.e.*, in the external environment, calls up in the mind a corresponding combination of two sense-perceptions within, *i.e.*, in the brain. Similarly the sequence of phenomena without, acting on the sensorial sphere, calls up a sequence of perceptions within. These are the rudimentary bases for the association of ideas. Observations on young children will reveal numerous instances in which their mental growth is seen to take place along the lines thus indicated.

Language—Co-operation in Brain Development.

The teaching and learning of language is, for the child, not a necessity but a convenience—it allows it to represent things by a common denominator. And the child is continually doing this. Thus the child learns to call its nurse Bi, and its term for to hide is tik. The nurse hides behind the curtain and the child learns to express it by the term Bi-tik. But one day it looks out of a window and sees the sun just getting covered over by a cloud; and the child now on this novel occasion cries out Bi-tik several times, pointing its finger to the cloud. Already the child had learnt to utter the two words together in the nursery for a special purpose; and now, under other skies the same expression re-appears, is re-uttered under the novel circumstances just noted. The somewhat crude similarity in the external sequence of events—a bright and conspicuous object, a dark curtain, and the one disappearing behind the other—combine to furnish the raw material for the building up of a new idea, new in one way, and yet not altogether new but having affinities (resemblances) to an item in the child's previous experience. The present author thinks that in this case the correct mode of regarding the problem of psychogenesis, is not to assume that the child in a deliberative and volitional and effortive way utters this proposition, but rather that this is effected *for* the child by the external logic of events. The *formation* of such rudimentary propositions first, and then their *extension* to things new and yet similar to and comparable with things familiar: these two processes are at the basis of psychological development in the child. Children see resemblances easily and they are exceeding anthropomorphic. On these known attributes are based also the system of education according to the kindergarten method by toys and objects, wherein the child not only sees and handles the thing he is learning, but, according to the anthropomorphic tendency of childhood endows it with human-like qualities and thus is led to take more interest in it. But according to the very laws of mind and brain growth a new and vastly important factor comes in now at this stage, viz., the *spontaneous or rhythmic activity of the brain;* whereby a proposition thus uttered as the result of an apposition of mental images, tends to be repeated by the child over and over again. This happens—as observations on children show—when the child is even away from the scene of action, and occupied otherwise or with nothing at all. In the absence, therefore, of a presentative stimulus, there is a quasi-spontaneous revival of the juxtaposed ideas in the mind, and their repetition over and over again—a re-visualising of things seen, a re-hearing of things heard, a re-vivifying of things previously experienced. The vividness of the child's memory for sensorial impressions, its free play and growth therein, and its fondness for gorgeous and fairy tales in which sensuous elements always preponderate, the vividness of its dreams, and of its terrors (night terrors, &c.) all combine to demonstate to us how the sensorial sphere of the brain is growing and developing in early childhood—both in association and intensification of mental images, and in their spontaneous revivals in the mind. Out of these there begin to grow conceptual ideas, and the child gradually learns to voluntarily *recall* its ideas. Our own view of the *rationale* of this portion of psychogenesis is as follows.

Further Mental Evolution.

When preconceptual propositions from frequent repetition are sufficiently organised in the mind, they get spontaneously recalled at times, without any obvious presentative external stimulus. The external logic of events is now no longer a *sine quâ non*, needed to revivify them. They arise spontaneously, they become *recalled* thoughts, *memories*. Thus, thoughts arise in the child's mind. The feeling of spontaneity which accompanies these thoughts of the child gradually get built up into the distinct recognition of the ego as the *empirical source and centre* of such feelings. Another and further factor, also, now enters into the sphere of psychogenesis. By so much as objects in the external world oppose the ego, constitute obstacles to its spontaneous and overflowing activities, by every obstacle overcome, every pain felt, so there is a clearer severance established between the ego and these others, its antagonists. The ego is thus built up empirically, and is gradually integrated and recognised as distinct from external objects. This is, therefore, the next gradual evolution from the sensuous and primitive rational stage of the child, viz., the stage of self-particularising, and integration of the ego. We shall trace the further growth a little way still. With the increased extent and multiformity of the environment with which the child comes in contact, with every increase in the amount of obstruction and antagonism encountered and overcome, the empirical ego grows in clearness and integration, and in its separateness from all else—the non ego. During this period of upward brain growth the more the ego—the growing highest nervous sphere—asserts itself, each new and added experience, each new activity and suffering but serves to sharpen the feeling of contrast between ego and non-ego, and to integrate the ego. Thus, true self-consciousness arises. In puberty and adolescence, it undergoes a second change and re-arrangement, from which it may emerge in the one case, arrogant, conceited, boisterous, coming close it may be even to the confines of adolescent insanity, in the other with its feelings and sympathies broadened, softened, and deepened, in which the purely self-assertive stage is largely built over with the higher state of self-sacrifice and altruism, the two necessary requisites for the higher development of human character.

Pathogenesis.

Here our exposition of the law of psychogenesis must stop, and we must now ask the question, if such be the story in brief, of the anatomico-physiological evolution of the brain, and such the law of psychogenesis, what, if any, is the law of pathogenesis under which the cerebral organization is deranged and destroyed, what *its* mechanism, and mode of action, and how far (if at all) can these be correlated with psychological and clinical observations? The whole cerebral organisation is not, of course, nervous exclusively. Non-nervous elements (protective, nutritive, &c.), also enter into the composition of the nervous mechanism, and these we must now briefly consider, to prepare the way for the last item of our inquiry—the Law of Pathogenesis. These non-nervous mechanisms of the brain we shall consider under the following headings, which are intended to be suggestive rather than exhaustive. They are:—(*a*) Protective mechanisms, and (*b*) Nutritive mechanisms.

(a) *Protective Mechanisms in the Brain.*

The whole complex of the nervous elements of the brain, both grey and white, stretching from external (pial) to internal (ventricular) surface is pervaded by a system of sustentacular elements, the neuroglia *fibre systems*. This is composed of a fine diffuse felt-work of fibrils radiating from stellate fibre cells (fig. 25), which are scattered throughout the whole cerebral mass. In certain regions they exhibit (as the present author has shown in two previous publications) a more condensed fibre feltwork, these regions are, especially (*a*) the surface region of the cortex, where a tangential sur-

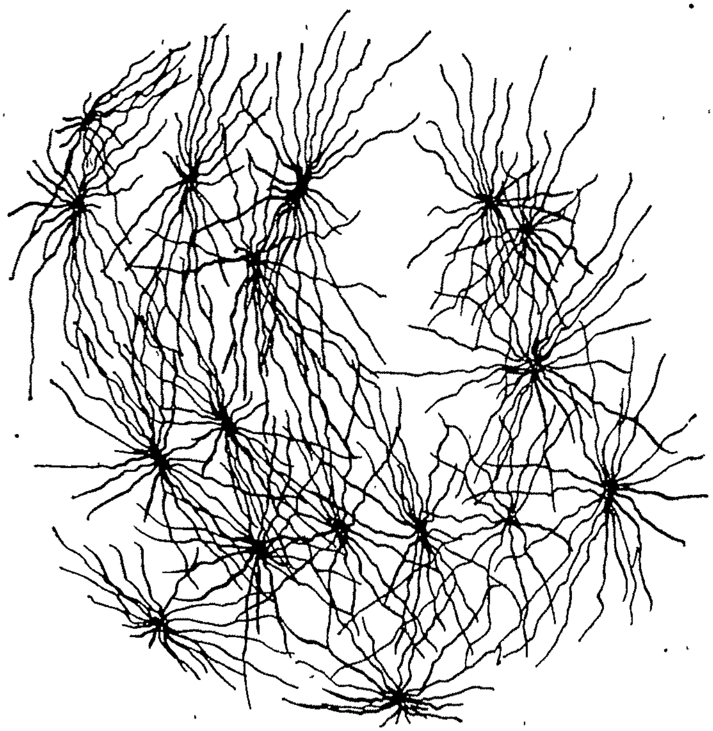


FIG. 25.

Several (17) neuroglia fibre cells from the human brain (medulla-oblongata) in the region of the olivary body, stained by Golgi's method.

face condensation system of fibres is found (see fig. 9), and (*b*) along the borders of the intra-cerebral vascular canals: perivascular condensation system. The author wishes to point out that each of these special developments has a profound significance in the economy of the brain. The surface feltwork system of fibres, mainly derived from the caudate neuroglia cells in this situation is an admirable protective mechanism, lying like a felted covering over the molecular layer, the first cortical layer, the one which contains the expanding, numerous and exceedingly delicate terminal protoplasmic branches, naked nerve fibrils and collaterals which form the very subtle and delicate nervo-protoplasmic plexus of this region. From the caudate cells in the deeper part of this felted layer another (a root) system of fibres pass in streaming tufts into the cortex, passing through the molecular, ambiguous, and sub-ambiguous strata into the *midst* of the region of long pyramidal cells, serving to support and sustain the nervo-protoplasmic and cellular elements in these regions.

(*b*) Similarly in the perivascular regions these fibre elements are also found developing into a special felt-work of neuroglia fibres limiting the vascular canals. The author may be allowed here to quote from his previous work on this subject.[1] The blood vessels of the brain have been stated to lie in channels excavated in the ground substance, so that an actual *space* can be demonstrated between the adventitia and the brain substance. This (extra-adventitial space) has been recognised by nearly all writers, including *Schwalbe* ("Lehrb. der Neurolog.," 1881), *Bevan Lewis* ("Text Book of Mental Diseases," 1889), *Obersteiner* (Translated by Hill, 1890), *Edinger* (*Zwölf Vorlesungen über den bau der nervösen centralorgane*, 1892), &c. "The blood vessels, as they dip down from the pia into the cortex, can be seen to be surrounded by a number of longitudinal fibres coursing in the ground substance next to the vessel wall, and following the vessel on its downward path. These fibres belong to the perivascular condensation system. This is constituted of at least three different morphological sub-types of stellate and elongate fibre cells. . . . These are:—(*a*) A number of elongate oval fibre cells (fig. 26), which, alike by their conspicuous appearance, and by the clearness of their staining, stand out in a wonderful manner. Their long axis is parallel to that of the vessels. They lie in the ground substance bordering the vessel, and in favourable specimens—when too many are not stained—their processes can be followed up and down along the vessel for enormous lengths. . . . . These cells lie at moderate distances from one another (fig. 26). . . . . Their fibres are slightly wavy in their course; they do not branch or anastomose, but run independently from cell body to their termination. The slightly oblique course of several of these fibres . . . . serves to produce the appearance of a loose open meshwork

---

[1] "Internat. Monatsch. fur Anat. und Physiol.," 1893, Bd. x., H. ii.

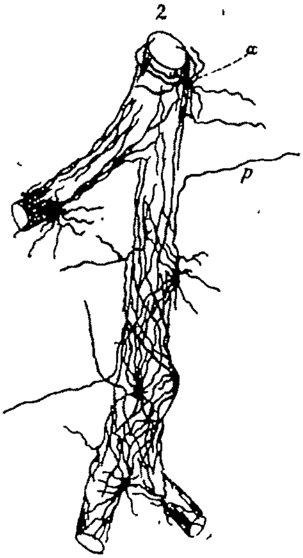


FIG. 26.

Blood vessel of the human brain, showing several neuroglia fibre cells surrounding it and forming a felt-work (perivascular system).

a. An encircling cell.

p. Perpendicular neuroglia fibre entering the sheath at right angles from a distant (extrinsic) cell—(Golgi's method).

lying on the surface of the vessels, the meshes appearing mainly elongated longitudinally, somewhat after the fashion of the meshes covering the lower part of a balloon (fig. 26). Intimately intermixed with these can be seen a number of cells which are disposed transversely to the line of the vessel, and are, therefore, best seen around transverse sections of the vessel. . . . . In medium-sized vessels they circle round the vessel for or ¾ the way, forming a system of embracing fibres. . . . . Besides the above two types of cell elements, which lie *in* the perivascular felt-work (intrinsic cells), the ordinary stellate neuroglia fibre cells, which are scattered throughout the general ground substance, also contribute a few fibres which enter perpendicularly into, and take part in the constitution of, the perivascular felt-work. . . . . . The majority of these extrinsic fibres—extrinsic because derived from cell bodies distant from and outside the felt-work proper—enter perpendicularly from without into the felt-work, and then turning off at right angles, course (within the felt-work) in a longitudinal direction parallel to that of the vessels' surface. A smaller number can be seen, however, running transversely over the circumference of the vessel, and after describing such a path—frequently a semi-circumference — over the vessel, emerge from the felt-work at the other side, and soon terminate in the ground substance . . . . Of the longitudinal fibres, but few are rigorously longitudinal. Apart from their wavy course many exhibit a slight obliquity, and can be often traced along the vessels' surface running in a *spiral* direction . . . . after the manner of the thread of a screw . . . ." For a full description we must refer the reader to our article in the journal referred to. Here we must content ourselves mainly with certain conclusions regarding the physiological significance of this sheath. "In the large vessels especially this sheath is of noteworthy thickness (fig. 27); a fact which must be nowise neglected in an investigation of the dynamics of the brain circulation on the one hand, and of the physics of the sustentacular and protecting elements (which support the nerve elements proper) on the other. For the interlacing fibres which unite into the perivascular felt work, must obviously form a structure which, on the one hand, would oppose considerable mechanical resistance to the *undue* expansion of the vessels in the cortex and white matter in the various brain areas, and, on the other hand, by its very texture and porosity would allow of the free passage and transudation of lymph

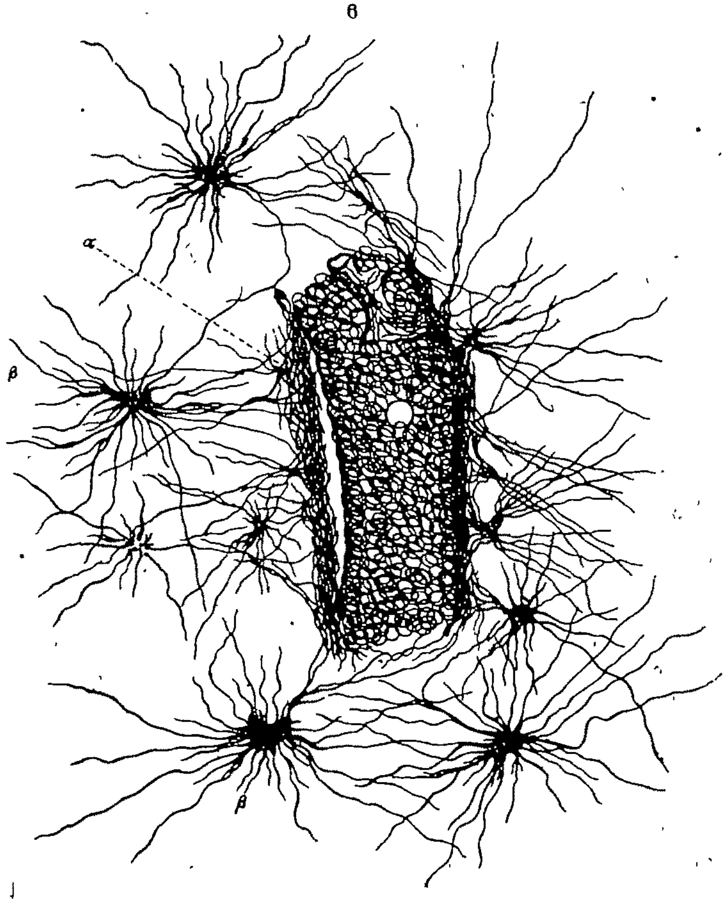


Fig. 27.

A large vessel, showing perivascular felt-work of neuroglia fibres; dense on the right side, less dense on the left side (*a*), where it is distinctively separated from the blood vessel by a space. (Human brain, Golgi's method.)

Note.—This and the previous drawing (fig. 26) are reproduced from the author's article on "The Blood Vessels of the Human Brain" (***Internat.*** *Monatsch. and Anat. u. Physiol.*, 1893, Bd. x., Heft. 11.)

and products of metabolism to and fro through its pores and meshes, and thus also allow of the interchange and diffusion of fluids and osmotic currents between the brain tissue and the perivascular lymph spaces. . . . . This function is further correlated to, and derives support from, the fact that the blood vessels of the brain have not only a *small* amount of muscular tissue, but are wanting in the tough adventitial elements (connective tissue fibre bundles of the areolar coat), which vessels in other organs possess . . . for the adventitia of the cortical vessels is simply, according to all descriptions, a tubular membrane, composed of a single layer of soft protoplasmic cells, while the bundles of areolar tissue, which are superadded to it in other organs, are absent in the brain cortex.

"The development of a felted sheath of neuroglia fibres in the ground substance immediately surrounding the blood vessels of the brain seems, therefore, to the author, of obvious significance, affording, by its structure, a protective mechanism for the limiting of undue local vascular dilatation, which might otherwise cause undue local pressure on, and consequent rupture, damage, and destruction of, the delicate protoplasmic processes, naked nerve fibrils, and collaterals, which pervade the grey matter (and to a less extent the white matter), while at the same time it admirably serves, by its texture and porosity, to allow of the free passage of lymph and nutritive products which enter into the fluid and general metabolism of the nerve centres." (*Loc. cit.*, p. 7-9.)

On the diffuse neuroglia fibre-system which pervades the whole of the general brain mass, we shall offer no great details; it is present in the cortex as in the white substance, and serves to support and hold together both nerve fibre-elements and cell-elements in general. The above is a *resumé* of the sustentacular and protective mechanisms we find elaborated in the brain, a series of structures which, considered in their whole, might well serve to show us how, in cerebral organisation, non-nervous elements have been evolved to keep pace with the nervous mechanisms proper in the race for life.

Ependyma Formations.

To complete the chapter of the neuroglia fibre elements we must now deal with that obscure structure lining the ventricles of the brain and continued along the central canal of the cord, a structure known by name as the ependyma, but of which *really* little was known, and a great deal, therefore, speculated about; amongst others, the hypotheses of Gaskell and Bland Sutton, which make this the remnant of a pre-vertebrate cephalic stomach and alimentary canal. The obscure and little-known ependyma was a fruitful ghost to conjure with, till the investigation of its structure and our real knowledge of it by the use of Golgi's method, and its phylogenetic significance as a *water vascular tube* permeating the central nervous axis in ancestral and acraniate vertebrata (as shown by us in our "Researches on the Pituitary Body and its relation to the Central Nervous System," *v. supra*) have combined to assure us of its significance, as one far different from the invertebrate gut hypothesis.

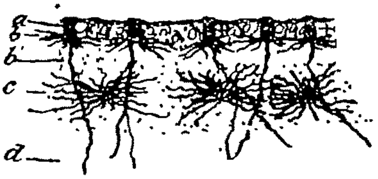


FIG. 28.

Ependyma of rabbit's brain (lateral ventricle).

a. Layer of epithelial cells.
b. Fine radiating basilar fibrils of the same cells.
b'. Coarse single basilar process.
c. Layer of special stellate fibre cells of the ependyma.
d. Transition region, passing into the extra-ependymal white substance.

Structure of the Ependyma.—This is shown in figs. 28 and 29; the one from the lateral ventricle (rabbit's brain), and the other from the spinal cord (kitten).

In the brain we see it composed of three layers: viz.: first, the epithelial cell-layer, in this case (fig. 28) but one cell deep. These cells are hexagonal in outline when looked at perpendicularly from the ependymal surface. In section (as in the figure) they are columnar or cubical. The free surface seems to be double-contoured: this is probably due to a beaded or striated appearance of the free border (? cilia). The deep surface gives off generally one stout basal process (*b*), and several radiating fine fibrils, which, by their interlacing, form a fine sub-epithelial intrinsic plexus. Beneath this is a third stratum (*c*) of stellate fibre-cells: these are coarser and more irregular in their processes than the ordinary stellate cell. They form a distinct layer (*c*); passing deeper still we pass into the extra-ependymal region; the cells of *c* forming the deep layer of the ependyma. Frequently between the epithelial elements and the stellate layer is a thin stripe, clearer and rather freer of fibrils.

Ependyma of the spinal cord (fig. 29) is composed of the following:

(*a*) Epithelial cells about three deep generally, surrounding the central canal. A careful study of their shape is important: some are short and squat; others more attenuated, the attenuated part frequently showing short angular spiny projections. These attenuated cells have the nucleus situated in the deeper (more globular) part. These cells also give off a thicker basal process, and finer and shorter basi-lateral processes.

A careful study of the figure will show that transitional forms are present, in appearance passing through the grades of the attenuated epithelial cell, and approaching the cells of the next layer, viz.: the sub-epithelial stellate cells. The mode of evolution which we have followed in all its stages in the spinal cord of kittens from the fœtus a week before term to the young one a month old, shows us that certain of the epithelial cells move or migrate to the deeper part of the epithelial layer the nucleus and main cell protoplasm retreating downwards. As this happens, the attenuated process which still reaches the free surface thins and eventually atrophies, while *pari passu* the basilar fibrils grow out and elongate laterally. Finally the primitive surface-process, and the thick long basilar process both thin down and atrophy, while the other fibrils grow out, and the cell now assumes the elongated stellate shape which is so characteristic. This morphological evolution is shown in B (fig. 29), the stages of evolution being 1, 2, 3, 4, 5 (fig. 29, B).

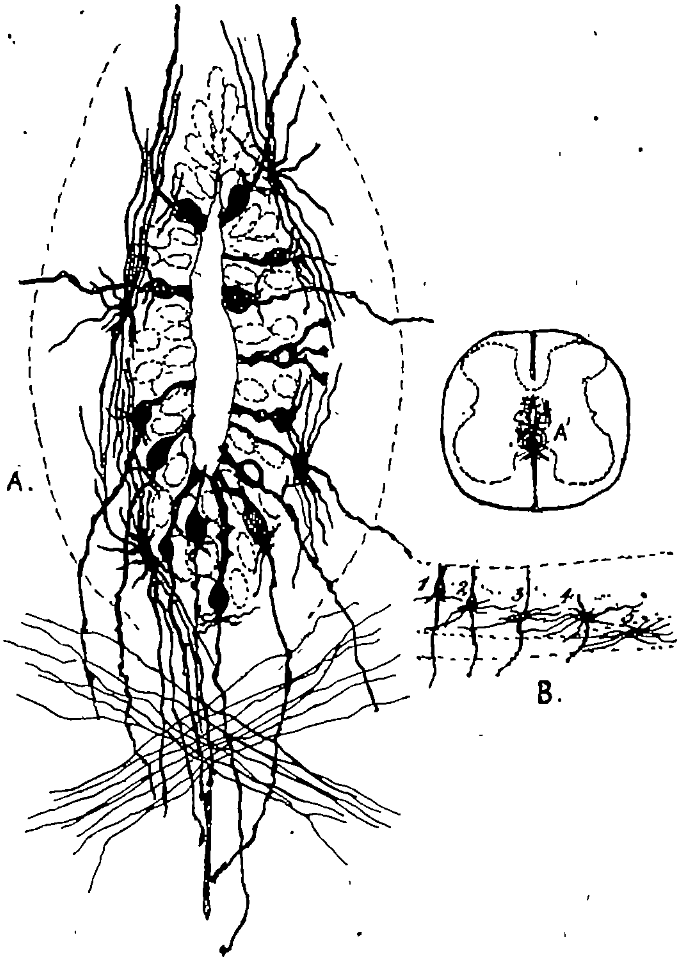


FIG. 29.

Central canal of the spinal cord, showing the ependyma. (From a young kitten's spinal cord, prepared by Golgi's method.)

A. *Ependyma* (shown on a small scale in the section of the cord, as A').

B. Showing the gradual evolution of the stellate neuroglia fibre cell from the epithelial cell, passing through the grades from 1 to 5.

These stellate cells, the last product of this evolution form circular strata surrounding the epithelial layer. There are minor points of detail which, though of interest, we must pass over. Our observations afford no support for the views of the invertebrate gut-theory. The elements of the ependyma are wholly and entirely epiblastic—every cell of it: derivatives of the primitive invaginated neural epithelium: of which some cells form neuroblasts, while others develop into these long epithelial cells whose tails (in the embryo chick as shown by *Golgi*, *Cajal* and others) reach to the pial surface. These cells are, therefore, called pia-ependymal cells; and as we have just seen, they give rise (by migration, and alteration of growth) to the stellate fibre-cell. These observations thus confirm the view we put forth in our previous publication (*British Medical Journal*, July, 1893), that the neuroglic fibre cells are epiblastic in origin. In the spinal cord the cells which have earliest migrated from the ependyma have reached the gray matter, and even the white matter outside it: some of these having migrated close to the very periphery of the cord, and there developed a number of fibrils. Two such cells from a kitten two days old we give in fig. 30; and yet another in fig. 31,

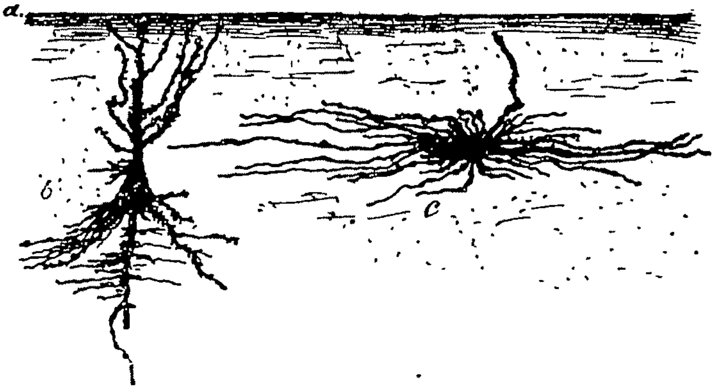


FIG. 30.

Two developing glia cells (fibre cells) from the spinal cord of a young kitten (2 days old). (Taken from the region of the lateral column)

a. Surface of cord.
b. Triangular fibre cell.
c. Oval (elongated) fibre cell.

to illustrate some of the varieties of these that occur throughout all the cord. Further description of these is needless.

(b) *Nutritive Mechanisms.*

We next pass to the nutritive mechanisms. These in the brain consist of—(1) Blood vessels. (2) Lymph channels. (3) Lymph secreting cells, and lymph absorbing cells. (4) Other cells and channels yet but little known.

Blood vessels. We propose to add certain new facts obtained by us in a research on their innervation. We find that it is possible to stain their vaso-motor nerves with Golgi's method. Starting from the carotid and vertebral plexuses we can trace them no further than the circle of Willis by anatomical dissection (using a lens). Do nerves accompany the cerebral arteries as they go off from the circle of Willis, and if so how far, and what is their ultimate distribution? Our observations on the kitten's brain show that bundles of nerve-fibres accompany the middle cerebral artery (the one specially chosen for our study), and its

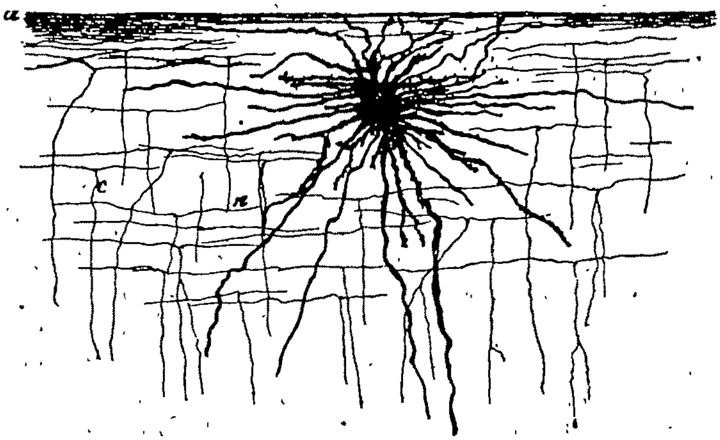


FIG. 31.

A developing glia fibre cell (stellate) from the cord of a kitten two days old.

a. Surface of the cord.

n. Nerve fibres running longitudinally (the section being longitudinal), and giving off—

c. Collaterals at right angles.

branches in the pia. These fibres run in tortuous and zig-zag fashion, and in the finer pial branches they can be seen to form a very fine (non-anastomotic) plexus of fibrils, lying between the outer and the muscular coats. From this peri-muscular plexus terminal fibrils issue which, running a short distance along the muscular layer either longitudinally or transversely, or obliquely, end in small spherules, little ovoid bulb-like enlargements, abutting against the muscular elements (cells). We have succeeded in tracing these terminals and their distribution to the finest pial branches, but no further. The intra-cortical continuations of the pial vessels have constantly failed to give us the least evidence of this peri-muscular plexus, which therefore—so far as our investigations go—we are compelled at present to imagine as stopping with the pial branches, and *not* continued along the intra-cortical vascular branches. The vaso-motor distribution—if our observations are confirmed—would thus seem to cease in the pia. Similar plexuses of nerve fibrils surrounding, and getting distributed to vessels in the choroid plexus, we have also been able to see, and in the new-born kitten's brain some of these nerve-fibrils are distributed to the epithelium cells of the choroid plexus.

In fig. 32, A is a small pial vessel with its peri-muscular plexuses of nerve fibrils and their terminals shown: B, a group of six cells from the choroid plexus, to which three or four fine nerve fibrils pass, ending by little free blunt points slightly swollen, and abutting against the epithelial cells. Are these choroid cells of the nature of secreting cells, and if so, are these the endings of secretory nerves distributed to these cells in company with the vascular nerve?

Lymph channels. As regards these we have nothing fresh to add to what is known.

Lymph secreting cells. Leaving out of question the choroid cells, we must mention in this connection those elements of the neuroglia which we have elsewhere described under the name of protoplasmic glia cells, and which we believe to be elements altogether different from the neuroglia fibre cells (*British Medical Journal*, July, 1893).

Quite fresh observations we have made on these with Golgi's methods confirm our previous conclusions concerning these

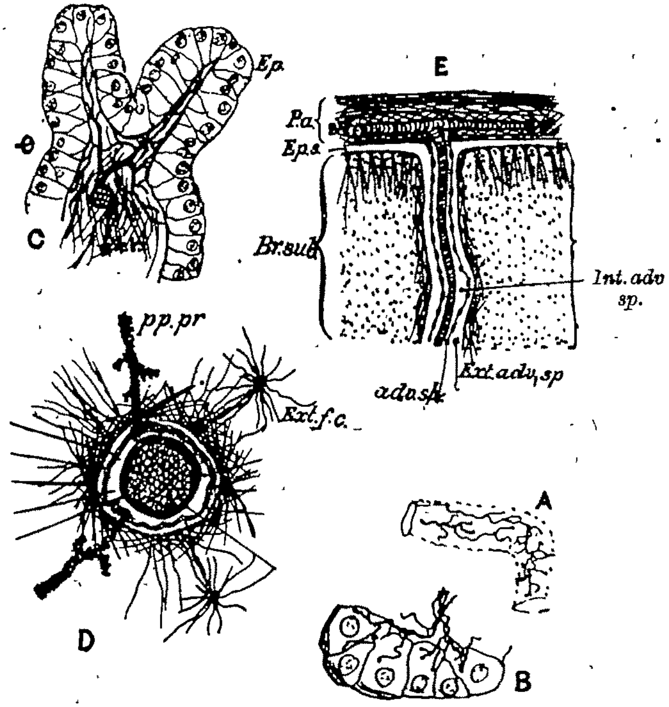


Fig. 32.

A. Perimuscular nerve fibrils (vaso-motor nerves) of a fine pial blood vessel of the brain.

B. Nerve fibrils distributed to the epithelial cells of the choroid plexus.

C. Portion of the choroid plexus.

Ep. Epithelial.

v. Blood vessel.

D. and E. To show the relationships of the vascular and lymph channels in the brain.

P.a. Pia-arachnoid.

Ep. s. Epicerebral space.

Adv. sh. Adventitial sheath.

Int. adv. sp. Intra-adventitial space.

Ext. adv. sp. Extra-adventitial space.

Br. sub. Brain substance.

cells which we have named protoplasmic glia cells. They occur abundantly throughout the grey matter in all the layers of the cortex, but are rare in the white substance.

"The protoplasmic glia cell has a distinct cell body, which is irregularly oval, frequently pyriform. Its various protoplasmic processes are of moderate length, they exhibit great variation in calibre, some being stout and coarse, and others exceedingly fine (fig. 33). These protoplasmic processes are also dendritic, a thing never seen in the stellate fibre cells. A most striking feature is the shaggy granular contour, as if a fine moss constituted the protoplasmic processes. . . . Further, by one or more of their coarser processes, the protoplasmic cells are attached to the perivascular sheaths. [Fig. 33 shows the cell with two such vascular processes each attached to the vessel by a conical disc-like expansion ("foot.")] We also stated at the time that the evidence of staining with *Golgi's* methods shows us a system of lymph spaces surrounding the cell body and its branches. Careful and fresh observations confirm us in this

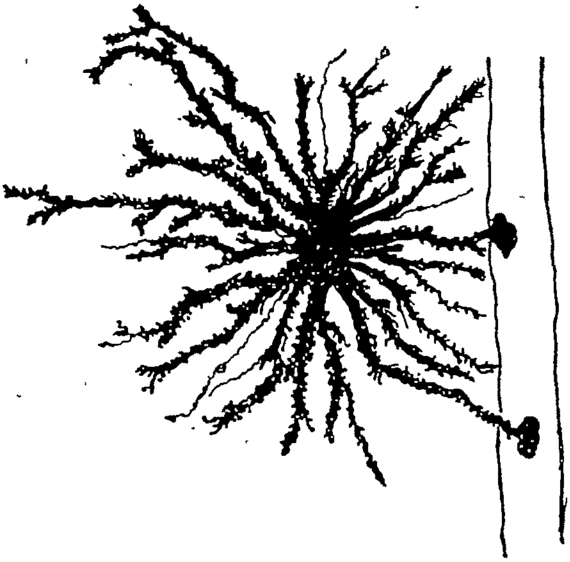


FIG. 33.

A protoplasmic neuroglia cell from the human brain (fourth layer of cortex), showing two expanded conical disc-like attachments to a vessel. Highly magnified.

opinion; viz., that there is an exceedingly fine system of canaliculi and lymph spaces surrounding the body, and dendritic processes of the protoplasmic glia cell, and directly continuous with the perivascular lymph spaces."

These protoplasmic glia cells are large and conspicuous objects. Besides these are a system of small glia cells—with little protoplasm, and cell body mainly nucleus—which surround the larger nerve cells as they lie in their pericellular sacs. These small cells have been frequently mistaken for leucocytes. They appear with remarkable distinctness in those cases of Golgi staining where the nerve cell bodies appear silvery white (unstained), against a dark grey or reddish brown background. They can then be seen within the pericellular sac as fine sharply defined nucleated cells, each with a little branching protoplasm, three or four such cells lying around the edge of the cell bodies especially of the large pyramidal and polymorphic cells. Their function is unknown. The former class of protoplasmic glia cells we regard as lymph secreting cells, the cell absorbing or taking up lymph from the brain tissue which it permeates, and discharging it *viâ* its peridendritic canaliculi, into the perivascular lymph spaces.

Other cells in the brain, of nature little known. We include under this heading, cells present in the molecular layer of the cortex (Cajal's cells); which that author is inclined to view as undifferentiated nerve-cells at one moment, and as spongioblasts (? sustentacular cells) at another. In the adult human brain we have not succeeded in positively seeing these, but they can be easily seen in the kitten's brain. Neuroglia fibre cells, which without care might be mistaken for these, occur of course in the molecular layer. Also certain round pigment-holding cells (? leucocytes).

Bases of Pathogenesis, i.e., the Insanities.

Having thus finished our survey of the total of elements—nervous and non-nervous—which enter into the cerebral organisation, and having so far investigated the life-history, growth, anatomico-physiological connections, &c., of the several kinds, we have endeavoured to show how, especially as regards the nervous mechanism, the upward growth is correlated to the facts of psychogenesis. We must complete our work, therefore, by now studying the reverse of this process, the method of pathogenesis ; to see where, how, and to what extent the progress and march of disease affects the nervous and non-nervous mechanisms, and if so, whether it is possible to correlate the pathogenic changes (neuro-pathology) with psychological and clinical observations (symptomatology). To illustrate these aspects of the problem we have chosen to consider the subject of the alcoholic type of insanity as one which from its clearness and definiteness of causation, its characteristic *onset* and *early history*, and the possibility of obtaining abundance of cases for clinical and pathological investigation, is a peculiarly happy one for the study of the law of pathogenesis in both its clinical and neurological aspects.

Alcoholic Insanity of Slow Growth.

Onset and early history. While in the majority of cases the onset and early history of mental disorganisation under alcohol point to extensive and wide-spread involvement of brain areas, it happens that in a certain number of others, these extensive and generalised changes are by no means the most prominent or typical feature, the change in these being more *intensive* and *specialised*, falling with peculiar vehemence on this or that sensorial, psychical, or psychomotor region, and comparable, therefore, to other local and intensive lesions like the focal epilepsies. It is, we think, in the latter type of alcoholic insanity we find the features which tie this class to the instinctive criminaloid and the epileptic individuals in whom the typical and peculiar morbid characteristics are also intensive and specialised. We have seen how, in the Jacksonian epilepsies, the primary focus of disease may be in some area of the psycho-motor, sensorial, or psychical sphere, and that the external manifestations vary accordingly.

So it is with our *early* alcoholic insanities. It may happen in *some* that the whole progress of the brain (mental) disease will have progressed in an *extensive and generalised* fashion, gradually lapsing into chronic weakmindedness and terminal dementia. During the period of lapsing into such dementia, or later, when the dementia is obvious, it will happen that these patients exhibit the most protean and multiform clinical symptoms—*e.g.*, senile excitement; emotionalism, epileptiform convulsions, cerebral softenings and paresis, apoplexies, acute hallucinatory excitement, &c., &c.: a vast sea of symptoms in which the flotsam and jetsam of the most varied psychical and bodily conditions are present; a state of affairs that has given rise to the adage that "to the alcoholic all things are possible." But before such an ultimatum is reached a definite and intermediate clinical history will have been gone through by the vast majority of alcoholics, the lines of march of these falling in certain fairly distinct and definite tracts, so that at the onset and in the early and intermediate stages it is possible to arrange the subjects of these diseases into certain definite natural orders. It is customary among neurologists, who, in the main, deal with the former kind of case, to state that the symptomatology of the chronic alcoholic is of the "extensive and generalised" kind, and such is the clinical type pourtrayed in our text-books (*vide* Gowers' "Diseases of the Nervous System," vol. ii., *sub. voce* Chronic Alcoholism). While these cases are by no means rare in our asylums, the majority of our alcoholic insane are those who belong to the other class in which the main morbid stress and evolution of symptoms is developing in this or that "intensive and specialised way." This latter is, at any rate, the predominant symptom, though careful investigation will in some cases reveal the presence also of the more generalised group of symptoms. The logical development of the symptoms in our opinion is from (*a*) the generalised and extensive to (*b*) the specialised and intensive, the real fact being that these first vary according to the personal equation, *e.g.*, heredity, stress of special sort, &c., under which the person lives. Having said this much by way of making our ground clear, we shall take up the feature in the onset and morbid evolution of the disease.

Generalised and Extensive Type of Onset.

The chronic alcoholic exhibits among the early recognisable symptoms, the following seven main groups:—

Generalised and extensive symptoms-complex.

First. Diminished power of recollection (amnesia), *i.e.*, of re-vivifying past mental images or ideas.

Second. Diminished power of attention and volition, *i.e.*, in sensorial or kinæsthetic spheres, including their interconnections.

Third. Diminished initiativeness and energy in conduct (correlated to one and two).

Fourth. Diminished muscular power; trembling (kinæsthetic sphere); muscular act (kinæsthetic centre) requiring to be reinforced by other sensory stimuli for efficient execution.

Fifth. Blunting of higher moral and ethical sense.

Sixth. Insomnia; loss of capacity for sleep and recuperation (*i.e.*, nutritive breakdown of the whole cortical sphere).

Seventh. Serious disturbance in the balance of cortical representations of *external* world and empirical ego, with melancholia and suspicion, delusions of persecution, and excitement, hallucinations, vague and gloomy feelings, and other pathetic emotional states, suicidal tendencies, passing into various chronic insanities.

We shall consider these groups of symptom groups *seriatim*:—

Clinical Symptomatology.

(*a*) *Diminished power of recollection* (amnesic type). Under the law of psychogenesis we had previously noted that the power of recalling past memories—of re-visualising things seen, of re-hearing things heard, and of re-representing previous mental images—imply a highly evolved cortical (mental) organisation; and that it has behind it a nutritive law of deep significance, under which "the external logic of events is now no longer a *sine quâ non* needed to revivify them. They arise spontaneously, they become re-called thoughts, memories. Thus, thoughts arise in the child's mind. The feeling of spontaneity which accompanies these thoughts gradually get built up into the distinct recognition of the ego as the empirical source and centre of such feelings" (*supra* p. 648.) This capacity depends upon the integrity, therefore, of the cerebral organisation in two regards, viz., (*a*) the integrity of the latest evolved and elaborated anatomico-physiological *connections* between the neurons which subserve it, and (*b*) the high nutritive elaboration, the high "nerve tension" and capacity for spontaneous discharge of the said neurons from time to time. Though these two are not separate, yet we shall consider them separately in order to be able to apprehend more clearly the pathogenesis of amnesia. In what respect does alcohol produce changes in these, viz., *first*, in the anatomico-physiological connections, or "field of conjunction" between the neurons, and *second*, in the *higher nutrition* of each individual neuron.

The results of our investigation with *Golgi's* method shows us that definite and recognisable changes occur in each of these respects.

Internal Pathology.

Changes in the anatomico-physiological connections (or "field of conjunction") between the neurons. Since the exact nature of such connections in health has been proved indisputably by the application of Golgi's method, it remains to inquire how and to what extent these are altered or destroyed, if at all. Our observations show that change of a very striking and unmistakable character occurs in the ultimate protoplasmic expansions and "contact-granules" situated upon them on the one hand, and in the ultimate naked fibrils (collaterals and terminals) which everywhere come into relation with such protoplasmic termini and granules on the other. Beginning with a softening and swelling of these contact granules, and also of the protoplasmic twigs on which they are situated (fig. 33), the earliest noticeable changes are a coalescence of these into small irregular "composites" of such, recognisable here and there (fig. 22 *a*) as a local coarseness. As the changes progress in coarseness and extent, they can now be more easily recognised (fig. 22 *b* and *b'*) as commencing moniliform swellings along the course of the terminal protoplasmic twigs. These are chiefly seen in the alcoholic brains we have examined with Golgi's method in the superficial parts of the cortex—molecular and sub-molecular plexus regions especially. With the further progress of the lesion these softened and enlarged protoplasmic masses form irregular botryoidal masses, mainly clothing the now irregularly bared

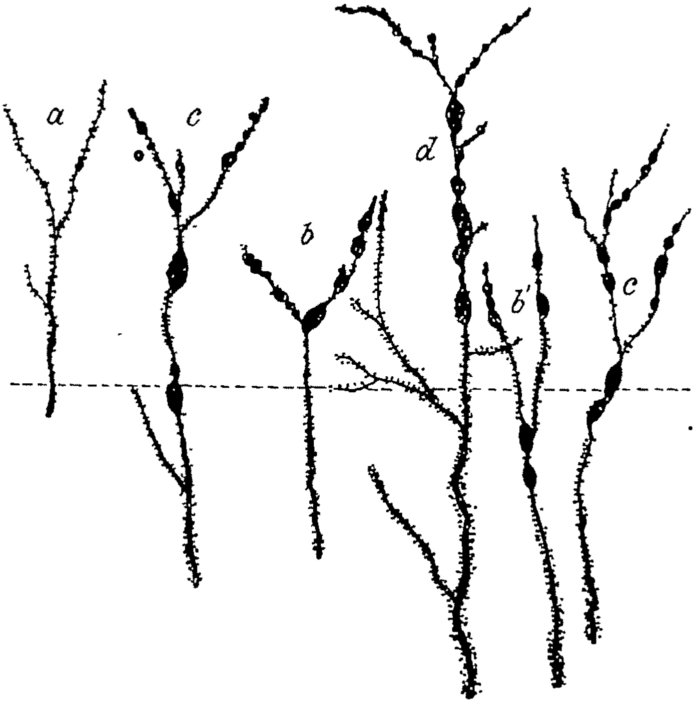


Fig. 34.

Terminal tufts and endings of the protoplasmic apical processes in the first layer (human brain cortex). Showing beadlike and moniliform swellings, coalescence of fine miliary granules in place, and loss of fine granulation in the most affected parts. (The dotted line marks the limit between the first and second layers.) (Alcoholic insanity.)

protoplasmic stem, which itself rapidly becomes softened, attenuated, and excavated (vacuolated) in places, till finally what is left is a mere ghost-like tattered protoplasmic stump, such as some of the cells exhibit in the last figure (fig. 34). These changes affect the apical expansions of both ambiguous, long pyramidal, and mixed pyramidal or polymorphic cells, and the chief stress of the lesions fall in the region of the molecular and sub-molecular plexuses, and in the sub-pyramidal plexus region. In the latter (and below) we come for the first time on the mixed pyramidal cells, and these show further changes, extending to the cell body itself, and remarkably striking in character. We are speaking of brains in which there is as yet no vascular blockings, and consequent softenings, for such we have purposely excluded from the present research, as the changes were too grossly secondary and advanced. The changes we now refer to are such as appear in this or that group of cells, when other

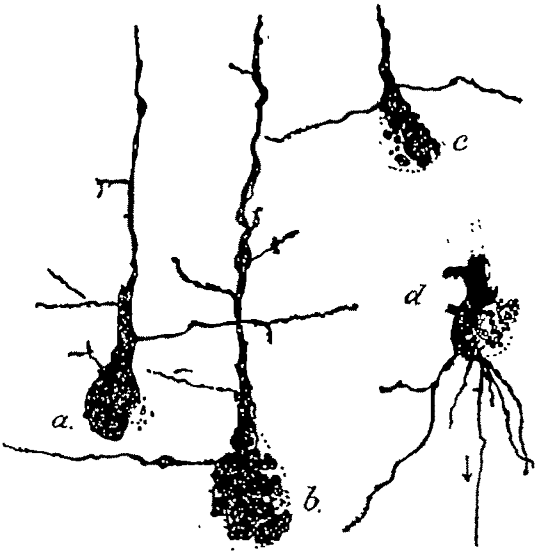


FIG. 35.

Four nerve cells from the human brain cortex (occipital region); situated in the deeper part (polymorphic layer) and showing various stages of disintegration (described in text). (Alcoholic insanity.)

cells in a part of the convolution a few millimetres off will appear but very slightly diseased. These changes consist in a gradual disintegration of the cell body (fig. 34); after the apical several of the basilar processes have considerably suffered. Here and there at the side of the cell body the protoplasm seems to get frayed, and, as it were, eroded or ulcerated away (fig. 34 *a*), the site of such being a local excavation of the cell protoplasm, with an adjacent little heap of *débris*. In other cells these changes will start from several points on the surface of the cell body (fig. 34 *b*), irregularly eroding and demolishing it, until but small fragments of the cell, mere ruins, with a little *débris*, are left in the pericellular sac (*c*). Such a cell, in which the destructive process has yet left its axis-cylinder and a few withered basilar-protoplasmic processes is seen in (*d*) (fig. 34). In other cases, again, the cell-protoplasm gets vacuolated from within, and the progress of this continues till its whole internal protoplasmic structure is channelled and tunnelled by such holes and seams of liquefaction. We do not mean to correlate early and recoverable alcoholic amnesia with the *advanced* destructive changes in the cell body. Rather we look upon it as associated with the early changes in the fine protoplasmic contact granules of the apical expansions which we have already figured and described. The lesions in these fine protoplasmic granules, however, represent but one element in the field of conjunction, and we must now refer to the other element, viz., the fine collaterals and terminals which, arriving from association, commissural or terminal fibres spread out in the three main plexus regions, viz., molecular, sub-molecular, and sub-pyramidal, forming the other element of the nervo-protoplasmic connections. These have to be carefully looked for with high powers, as, owing to their extreme delicacy, and their paleness of staining with silver chromate, they are else liable to be overlooked. A careful search, however, will soon reveal the fact that these are extensively changed and altered. The changes we have noted are mainly of two *kinds*, external and internal. *External changes:* These refer to the outline and contour of these fibrils. In health they are fine, delicate, smooth, and clear-cut in outline, like a thread of silk, and stain *internally*, with a uniform clear coffee-colored tint. In disease these become *granular* and *wrinkled* in outline, as though the firm smooth consistence of the fibre underwent a granulation with softening, and lost its cleanness of contour. Here and there, too, small irregular swellings are seen. *Internal changes* : These are very distinct in many specimens, and can leave no doubt in our mind that chemical alterations are taking place within the fibril. The most striking feature is discontinuous and uneven staining. The fine and delicate collateral or terminal fibril, which stained (if it stained at all) of a uniform clear coffee tint, is irregularly stained along its course, in some parts the pallor and want of staining almost indicating that the fibre is broken into separate fragments, till the most careful focussing and search will reveal the unstained intermediate parts. These streaks of discontinuous and irregular staining are so remarkably frequent and conspicuous in several specimens we have examined, that we have no doubt they represent what is a real pathological change going on within these naked nerve fibrils.

Correlation of Clinical with Pathological.

These two sets of changes in the naked collateral and terminal fibrils (internal and external) constitute, to our mind, the other element of earliest and recognisable change in the field of conjunction, whereby the pathways for the passage of nervous excitations from neuron to neuron, and from one complex group of neurons (nerve centre) to another, is thus rendered difficult or impossible. Both these sets of changes in the field of conjunction are, as far as the present author is aware, only recognisable with the use of Golgi's method and its modifications, the author having used for this purpose his own modification, published in the *British Medical Journal*, March, 1894. Our work in this line leads us to the view that it is here, in the changes in the anatomico-physiological junction regions, in this field of conjunction, we find the earliest dynamical changes, and that these represent, on the psychical side, the diminished capacity of the neurons to be excitable. to presentative sensorial stimulations (the equivalent of the softenings and obliterations of the protoplasmic contact-granules), and a diminished permeability in the pathways of nerve currents issuing from one neuron by its nervous process and its terminals to another neuron in the cortical area, the psychical counterpart of which would *be a slowness in the arousing of associated images, and delay of reaction time*. Finally, as regards the earlier and subtler nutritive changes within the cell body and nucleus. The physiology of nutrition is in the nerve cell elaborated to a high degree, and each nerve centre of this or that part of the central nervous system has its own intrinsic nutritive rhythm. In the alcoholic brain, the early and subtler changes affect, not merely the field of conjunction (produce *dynamical* effects *as far as the reception and transmission of nerve currents are concerned*), but the *trophic or nutritive focus* (cell body and nucleus) of each individual neuron. The *advanced* trophic changes, especially as shown in the polymorphic elements, we have already described and pourtrayed. The cells in this region are also favourable for the study of the *commencing* trophic changes in the neuron. The methods we have used for this purpose are four in number: (*a*) sublimate and alcohol fixing, and staining with Toluidine blue; (*b*) frozen sections, stained with aniline blue-black; (*c*) Nissl's methylene-blue method; (*d*) Golgi's method. Each of these helps to supplement the other.

Commencing trophic changes in the cell bodies and nucleus. We are of opinion that these also are definite and recognisable, and further, that in certain respects (viz., as elucidated by the aniline blue-black method) these have been abundantly described and figured by Bevan Lewis ("Text-book of Mental Disease," 1890). For our own part we would add certain details obtained by our investigations with the three other methods named, viz., the Toluidine, Nissl, and Golgi methods. The first and second of these methods are useful for showing certain fine intra-cellular and intra-nuclear changes which the aniline method does not help to show, while the third enables us to apply a certain test to identify what fragments of nerve-protoplasm still remain. Fortunately for us, not all the cells show equally advanced changes in the alcoholic brain, for while some show moderately advanced intra-cellular changes at once recognisable as such, others (neighbouring cells) on whom the stress—for some reason or other—has not fallen so severely will show us appearances consistent with health, or only faint indications of a commencing departure from the normal. This graduation of pathological changes helps us greatly, for it offers an intrinsic control method of distinct value. Keeping this in mind the earliest changes we see are: (*a*) Deeper staining, of cell body and protoplasmic processes (with aniline, Toluidine, and Nissl's method). (*b*) Swelling and softening (indistinctness of outline) of the intra-cellular chromatin rods, and increased intensity of their staining (with Nissl's method). (*c*) Thickening of intra-nuclear reticulum, and increased prominence of nucleoli (with Toluidine method). (*d*) Increase of the normal golden cell pigment which is present at or near the basal portion of the cell (with all three methods). We are not as yet in a position to state which of these four changes occurs first, or whether any two or more of them occur simultaneously; but certainly they all occur in the *earliest* stages, when the contemporaneous changes in the finest nervo-protoplasmic regions which we have before detailed occur. To us these four points mentioned indicate two things, viz.: (*a*) that there is increased functional activity in the cell body, and (*b*) that this is productive of a degradation in the form of nerve energy, the physical accompaniment of which is a softening and alteration of the blue chromatin rods and granules, and their gradual replacement by yellowish pigment. The increase and progress of pigmentation is the equivalent of the onset and progress of *degradation* in the functional activity of the cell—the replacement of living protoplasm by non-living pigmentary product as in senile decay and in chronic epilepsy: here (in chronic alcoholism) it is attended with the other intra-cellular changes of a more subtle kind, viz., intensity of staining of cell protoplasm, and swelling and softening with increased staining of the intra-cellular chromatin rods and granules, the reservoirs for the building up and storage of nerve energy under high tension, *i.e.*, the pabulum of the cell. The earliest lesions, therefore, of alcoholism are of a nutritive and dynamical type, dynamical in so far as it affects the protoplasmic granules on the one hand and the nerve collaterals and terminals on the other which enter into the constitution of the nervo-protoplasmic plexuses (fields of conjunction) in the brain; and nutritional in so far as it affects the protoplasm and nucleus of the cell body and therefore interferes with the higher evolution and elaboration of nerve energy, and especially with the quasi-spontaneous or voluntary vivifications of former memory images which is the peculiar property of the cortical neuron during its period of increased functional activity.

But this is not the only result of the alcoholic lesion. Another outcome of it is the next group of symptoms.

Clinico-Psychological.

(*b*) Diminished faculty of *attention* and *volition*.

Here there is a slightly deeper involvement we think. In the mild amnesia we have considered there is the loss of *re-vivification* or re-rousing of past experienced mental images and states; here, even when the objective images are present, the capacity of receiving and registering them in the mind is diminished. On the psychical side this is, in other words, diminished power of attention. It may be that in one case this is mainly limited to the visual, in another to the olfactory, in a third to the kinæsthetic, or in a fourth, &c., to the auditory, &c., sphere, and that in other cases it extends to all these. The faculty of attention is an early one in the infant, and we think it is simpler in character and grade than volition. At least two sensorial elements enter into the composition of attention, viz.: (*a*) the special or local sense which is being active; (*b*) its special kinæsthetic auxiliary. The mode of evolution of each special or local sense has been a twin evolution, a special kinæsthetic element having entered into it, and got incorporated with it. With sight there is the representation of the movement mechanism, which is concerned in *fixing* an object seen, in bringing it into the field of clear vision before it can be the special object of attention. With it comes *accommodation*, which is also brought to bear on the object. Besides these bin-ocular and intra-ocular kinæses a head movement may also enter into the primary constitution of attention to a visual impression (turning the head towards it), *cf.* also auditory attention and movements of the ears. This is the primary kinetic reaction, appearing early in the animal kingdom, and early in the child. It is fore-runner of and pre-requisite for attention.

So in hearing there is the external ear movement and the internal one (tensor tympani), these kinetic elements entering into the composition of auditory attention. So with smell (nostril and respiratory indrawing as in the child on its mother's breast, &c.). And with touch or tactile there is a parallel incorporation of a movement sense of the organs of touch (the fingers, hand, &c.).

Attention is thus analysable into a compound or complex of at least two sensory elements; therefore it is a higher sensory state (psychical state), and is the psychical concomitant of nervous action in such conjoint sensorial sphere. The feeling of effort enters into both attention and volition, and this has misled some to speak of the act of attention as an essentially "motor" process. But this is due to a little confusion of ideas. The sense of sight, of hearing, the tactile sense, the muscular sense, &c., each of these has its appropriate auxiliary muscular organs, the function of which has been to produce a movement in the peripheral sense organ. This has been so constant a thing that from frequent repetition their organisation has become perfect, until in man the motor action is habitual and follows the sensation. Thus according to our view every sensory organ has its auxiliary muscles which by their aid help to reinforce its function. The cycle of events is: (*a*) Stimulations of cortical sensory centre = consciousness of sensation. (*b*)

Movement following, whereby the external object is brought into the *centre* or focus of the field of consciousness by pricking of ear, fixing with eye, &c. (*c*) As the result of. (*a*) and (*b*), stronger arousal of cortical sensory centre by the re-entering sensory currents both from the *sensory organ* and its *auxiliary muscles.* Such re-entering of auxiliary kinæsthetic currents serves to *reinforce* and increase the *functional. activity* of the sensory centre. The psychical side of such intensification of the activity of the sensory centre is attention. Hence we cannot localise the faculty of attention in any one "area" of the brain (as *e.g.*, Ferrier, in the pre-frontal lobe in monkeys). It accompanies, and can accompany the activity or any of the specific centres of sensation, whether visual, auditory, tactile, gustatory, olfactory, or kinæsthetic. There is no special centre for the faculty of attention.

Now the above heightening of the functional activity of a sensory centre has for its object some act immediate or future on the part of the organism in response to the sensation which is now distinctly felt. It may be now one, or it may be at another time other act, according to the dynamical (anatomico-physiological) connections possessed by the sensory centre which is in a state of such increased functional activity.

During the condition of attention there is thus not only an increased functional activity in this or that sensory centre, but an overflow or discharge from that centre to *others* along definite routes, or diffusely all over its borders. Where a voluntary act or movement is one of the outcomes, the neural discharges consist of (*a*) primary sensation, focussing-reflex, and attention ; and (*b*) discharge from such sensory centre, (*c*) with resultant excitation of other sensory or psychical centres, arousing feelings and mental images (ideas), or of a kinæsthetic centre, in the last case evoking a more or less obviously special movement. The sequence of events comprise, therefore, (*a*) arousing a sensory centre to attention ; and its discharge, along (*b*) a tract to (*c*) a kinæsthetic centre ; followed by an appropriate movement to its completion, or, in psychological language, *first,* perception; *second,* apperception and attention; *third,* strong revival in mind of the act to be performed; *fourth,* execution of the idea.

Attention thus belongs to the sensory side; volition to a specialised and intensive discharge therefrom to the kinæsthetic sphere. Volition is thus a development from attention, and passes on to execution; it is thus the passing from *attention* to *execution:* in the brain it overlaps the psycho-motor sphere on the one side, and the sensory on the other: its region is, therefore, the *transitional* or association system one between these two.

On the anatomico-physiological side we think the mixed pyramidal or polymorphic system to chiefly represent this association region, partly on comparative and developmental grounds, and partly from pathological considerations. The gradual historical development and elaboration of this system in the mammal, till it attains its acme in man, and the lateness of this lower cortical organisation to complete its growth in the new-born and young indicate that this "accessory association system" of the brain is the chief structure which subserves the higher psychical functions, and especially volition. Further, of all the various cell systems involved in chronic alcoholism, it is the one in which the changes—especially the trophic ones—are most advanced.

Pathology.

We therefore correlate its special implication and various before-described phases and early change with the special neurasthenia of the alcoholic, including the next group of symptoms on our list—diminution of initiativeness in conduct, and laziness with blunted moral and ethical sense behind these.

"Motor" Symptoms.

Coming next we have a definite group of symptoms, viz.: (*a*) *loss of* muscular power, with tremor on exertion. We regard this special condition as due to lesions in the kinæsthetic area, affecting both the dynamical and nutritive parts of the nerve apparatus; *i.e.*, both nervo-protoplasmic connections and cell bodies. Indeed, this Rolandic (or movement) sphere is the commonly used one for its study because the cell elements are large and easy, therefore, for study in this situation; and perhaps because the further progress of the lesions are apparently more conspicuous and striking here (*e.g.*, atrophy and wasting of convolutions: œdema and thickening of pia-arachnoid, &c.). We have seen in studying the development of the Rolandic region in the young how the increased growth and extent of collaterals from the descending axis-cylinders of the ambiguous and pyramidal cells, and their coming into further contact with the protoplasmic expansions of the lower and larger pyramidal cells constitute a special intrinsic mechanism for the consolidation of movements. These collaterals conspicuously suffer, viz., in the Rolandic area, and to this we attribute the tremor and unsteadiness (dynamical disturbance) in movement. Apart from this tremor there is a true *weakness*, as shown with the dynamometer, which may amount to a paresis of 20-40 pc. below health.

Pathology.

We correlate this with the commencing nutritive degradation of the nerve cells which discharge down the pyramidal tract, inciting thereby the motor cells of the cord. It is, we think, a phenomenon parallel to the muscular weakness of neurasthesia when tested with the dynamometer. This combination of tremor and weakness are the basis for the dyskinæsis of alcoholism.

The reinforcement these cells require, even to enable them to act continuously for a time, should be noted. The alcoholic holding a ball or jug in his hand will often drop it if he be not looking—fixing his attention—on it. For the normal tactile and kinæsthetic ascending currents arrived at the brain, have to act on pyscho-motor cells of but low energy which may flag. By looking at the object (attention) the sensorial visual excitations arrive after passing the visual centre, to the hand centre, and *reinforce* the volume of the sensory exciting currents which keep up the functional activity of the psycho-motor cells. The phenomenon here is also analogous to that of subminimal stimulation of the cortex, which, though of itself failing to evoke movement from the kinæsthetic focus, will do so if the paw be first stroked gently a few times, and then the electrodes applied: The centre thus slightly roused in its excitability now just reacts. Instead of the paw the stimulus of some associated area (*e.g.*, *smell*) will also serve the same purpose, viz., heightening the excitability of the kinæsthetic centre. (In this connection note also Belmondo's experiments on the spinal cord motor cells associated with stimulation of the posterior roots, *Archiv. Ital. de Biol.*, 1891, part i.), which also fall into a similar category.

Clinical.

(*b*) *Insomnia.* This is another of the early alcoholic symptoms. How far this may be due to habit is difficult to say. But its injurious effect on the brain is certain. We know from physiological experiments that cell elements, whether secretory or motor, exhibit physico-chemical changes during activity: *e.g.*, electric changes and changes in intracellular structure. When pushed to the limits of fatigue, these changes in the *secreting cells*, the *muscle fibre*, or the *moter end plate* of the nerve are not too difficult to recognise. A pseudo-sanctity which has hitherto surrounded the nerve cell and sought to exclude it from the circle of these metabolic changes is now breaking down, and of late years the experimental work of cerebral thermometry (Mosso), and of the microscopic changes in nerve cells accompanying functional activity (Hodge and others) have gradually brought a series of positive facts to displace the old superstition.

Pathology.

Hodge's work in this respect, following on the older observations of Sadovski and others, shows that in the cerebral cells of the honey-bee, swallow, pigeon, and sparrow, such recognisable changes are found. To summarise them, he finds (*a*) changes in the nucleus, viz., shrinkage, and crenation of outline, loss of fine intra-nuclear reticulation, and tendency of the nuclear chromatin to stain more darkly than during rest. His experiments were extensive, carefully and laboriously done with abundance of control material, and the results obtained are regular and very uniform. Besides these intra-nuclear changes, there are (*b*) the alterations in the cell protoplasm: these include shrinkage in size of the cell with (in the case of the cerebral cells) enlargement of the peri-cellular lymph space, commencing vacuolation in the cell protoplasm, and diminished staining reaction of the fatigued cell. The present author having been favoured with the actual microscopic specimens from Dr. Hodge, can entirely concur in the above statement of facts as borne out by the preparations. This subject has also a further interest owing to the researches of Dr. Batty Tuke, who, with Dr. Mann, has investigated the changes underlying fatigue in the bipolar cells of the retina (second layer), and in the occipital lobe of the rabbit in connection with visual stimulation, as well as the changes in the cells of cervical ganglion also, during fatigue induced by stimulation of the sympathetic cord lower down. The specimens and micro-photographs which illustrate their research (yet unpublished) leave no doubt that a series of changes parallel to those described by Dr. Hodge are present beyond doubt. Their conclusions I present in Dr. Tuke's own words: "In the unstimulated cell the nucleus stains lighter than the protoplasm; the first effect of stimulation is to reduce the staining of both to the same degree of intensity; as it is continued the nucleus darkens, but remains lighter than the protoplasm; then the nucleus becomes distinctly darker, and begins to get deformed and crenated; and eventually a condition is produced which may be spoken of as collapse, nucleus and protoplasm losing all power of taking on stains." The third stage, in which there is the conspicuous dark staining of the nucleus, as well as of the cell protoplasm, and in which the nucleus itself begins to get deformed and crenated is stated by Dr. Batty Tuke to be the "limit of functional activity changes." Experimentally it has been reached after eighteen hours' continuous excitation of the cell, and marks the borderland where the whole pabulum or nutritive material of the cell body is used up, and yet leaves the organ in a condition admitting of reconstruction (recuperation). A fourth stage, according to the same observer, marks the pathological, the condition of actual or commencing vacuolation and disintegration within the cell protoplasm "from which it is doubtful whether full recuperation can be obtained." The consensus of results obtained from the work therefore of Heidenhain, Langley and others on glandular secreting cells, of Kodis, on cells of the epidermis, *Sadovski* ("On the Changes of Nerve Centres caused by Peripheral Irritation," 1889), and *Madame Ternonski* ("Changes in the Spinal Cord due to stretching of the Sciatic Nerve, &c.," 1885), on spinal ganglia and the cells of the spinal cord, and the more recent work of Lambert and of Vas on the sympathetic ganglia, and of *Hodge* on nerve cells ("A Microscopical Study of Changes due to functional activity in Nerve Cells," 1892), and of Drs. Batty Tuke and Mann (on the same subject, 1893-4) form a growing body of definite and well ascertained facts which the present author thinks will have the profoundest bearing on the scientific basis for practical treatment in neurasthenia, insomnia and fatigue, with commencing breakdown, which mark the milestones along the high road to the ultimate development of the various alcoholic insanities, the melancholias and the various other protean brain and nervous maladies which are so abundant in our decade.

And this leads us now to the next group of symptoms-complex in our general consideration of the alcoholic group of the insanities, which completes our general survey, viz.:

Clinical.

Melancholia with suspicion, vague dreads, mixed with phases of momentary excitement, and finally of acute hallucinations and delusions, maniacal excitement, delirious conditions, and epileptic fits, from which, if relief is obtained, a permanent residuum of weakmindedness is left behind, or of chronic mania or systematised delusional insanity. The earliest in this group is melancholia, with vague and general suspicions, intensified at moments with distinct hallucinations of the senses. It is at this stage that most of our asylum alcoholics come under observation, having at home passed through the previous six stages. This, the seventh stage, is one of bad omen. It indicates not only a serious progress of the mischief beyond the limits where, according to Batty Tuke, repair is possible, but is very often the beginning of a downward progressive stage of peculiar malignancy, most unsatisfactory to treat, and very disheartening in its sequelæ. Of course all parts of the higher cerebral organisation have not suffered equally in these cases; the clinical and psychological indications they exhibit prove this. But though the patient be placed under good hygienic conditions, with rest, abundant food, the absence of worry and anxiety, and the administration of sedatives to procure sleep, of massage for the body tissues, and even of intestinal asepsis, in the vast majority of cases the capacities for recuperation have been passed, and their march and progress is towards systematised and chronic insanity.

Pathology.

Considerable extents of cortical tissue have been damaged beyond repair: points of softening and breaking up in the molecular and sub-molecular nervo-protoplasmic plexuses, and a more notable involvement in the sub-pyramidal region (polymorphic system) with pigmentary and fatty degenerations of nerve cells, cutting off and complete degeneration of one or more entire basilar protoplasmic processes or of the whole apical process, and similarly to crown all a vascular disease (endo-arteritis and fatty changes with narrowing of lumina of cerebral blood vessels) with *its* sequelæ, viz.: imperfect nutrition of the brain tissue, thrombosis and focal softenings which complete the picture of progressive rottenness and disintegration of brain tissue which can never be recovered from. This stage is associated with softenings and swellings in the neuroglia elements themselves, with *vacuolation* of their cell protoplasm, with pigmentation of the cell body, with swellings of the processes (fig. 36), and the abundent formation of so-called colloid bodies in the brain. Our observations show us that in a considerable number of cases these latter structures, viz., "colloid bodies," are derivatives of the neuroglia fibre-cells, as revealed by the method of staining with silver chromate. They are to be met with in all sizes, from the smallest recognisable speck situated on a delicate neuroglia fibre to a considerable swelling or series of beaded swellings occurring along the course of the same neuroglia fibres. As to their origin in medullated fibres from swellings and spherules of the myelin substance, our observations give no indication (negative evidence), but it leaves no doubt in our minds, in so far as the sharpness and precision of staining with Golgi's method leaves no possible

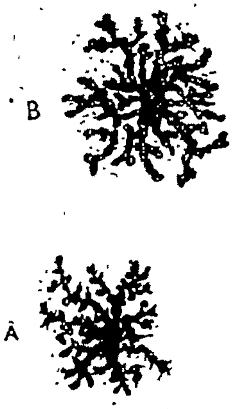


FIG. 36.

Two protoplasmic neuroglia cells of the human brain showing two stages of change.

A. Commencing beaded-swelling of the protoplasm.

B. A more advanced stage; the protoplasm in coarse irregularly-beaded botryoidal masses, with loss of fine mossy-granulation. (Alcoholic insanity.)

ground for doubt, that at least one source of their origin is the neuroglia element—the fibre-cell. In the surface condensation system of neuroglia fibre, in a region where the medullated fibres are absent, these bodies can be recognised in all sizes from the minutest to the largest met with. Is it not singular that according to the view of the origin of these bodies from myelin, these bodies should be rare and conspicuous by their absence from the very heart and centre of Exner's plexus, where medullated fibres abound, whereas when we pass more superficially through the "clear zone" (see fig. 22), and enter into the substance of the tangential plexus of neuroglia fibres, that here, in a region barren and devoid of medullated nerve fibres, but abounding in neuroglia fibres, these colloid bodies should be abundant? We have also noted their presence in the ependyma in connection with the special layer of stellate cells in the subepithelial region, these bodies being undoubtedly in this region caused by swellings along the processes of these stellate fibre cells. With alumhæmatoxylin we have confirmed the statement of Obersteiner that they stain a fine blue colour, and their deep violet reaction when treated with iodine, followed by dilute sulphuric acid. With Ranvier's picro-carmine they also stain darkly, but not with aniline blue-black. Chemically we regard them as coming closest to the eleidin granules discovered by Ranvier in the epidermis. We have now in the briefest manner to touch upon the specialised and intensive lesions in chronic alcoholic insanity to finish our general survey of the law of pathogenesis.

Special Heredity.

Specialised and Intensive Pathological Types of Alcoholic Insanity.—The subjects of these have a neurotic heritage, epileptic, intemperate, and criminaloid. The early and severe implication of certain regions of the cortical organisation in these betrays to us a significant fact, that the vice is in the nerve cell; the cell is the selective agent, so to speak. Under stress of another kind, *e.g.*, worry, sexual excess, &c., the result would have been a form of downbreak (disorganisation) of a similar sort (*e.g.*, melancholia and delusional insanity) to what it would be under alcoholic stress. We must recognise the intrinsic hereditary vice of organisation of the nerve cell; giving to alcohol, syphilis, sexual excess, worry, &c., or other external facts, its subordinate position as an element of *stress* which, whether acting directly as a chemical poison, or indirectly, *viâ* the peripheral nerves, is but an auxiliary in bringing out the intrinsic cerebral defect. The intrinsic vice of organisation is within, and requires but little stress of circumstances to reveal itself. In these subjects the "generalised and extensive" group of symptoms before mentioned is rapidly passed over or abbreviated, and the special and intense insane involvement early makes its appearance, viz., melancholia with persecutory delusions (two-thirds of the total number of cases) with or without ideas of grandeur, varied by outbursts of acute excitement (mania), in which acute hallucinations may play at times a causal part. These hallucinations and delusions may be *aural, visual, olfactory, cutaneous, gastric and abdominal, genito-urinary*, &c., or several of these combined, in fact the most protean and multiform that could be imagined, and having affinities with the more rapidly evolving G. P. on the one hand, and the more slowly developing paranoiac on the other, and equally with those of hopeless prognosis.

We have now concluded our brief survey of the law of pathogenesis, which in a future publication we intend to deal with in greater detail than is possible within the limits of the present paper. We have seen how the onset and early course present all the phenomena comparable to fatigue conditions of the nerve cells, and that when these nutritive changes pass a certain limit, the manifestations of insanity commence. We have seen also how, with the involvements of the delicate collateral and terminal fibrils and protoplasmic granules which enter into the dynamical constitution of the cortical centres, this general diminution of brain power will be accompanied by sensory, psychical, and kinæsthetic clinical symptoms of a very distinct and definite kind, and how with the added vice of hereditary taint the law of psychogenesis finds its rapid and full realisation in the various delusional and melancholic states, with alternating phases of gloom and excitement, which passes on by natural evolution to the more hopeless and helpless grades of insanity.

From the whole body of the foregoing research and discussion, long and tedious as it has been, the following conclusions are therefore drawn:—

General Conclusions.

First. The doctrine of localisation of functions in the brain is the basis which renders a study of the psychology and pathology of insanity possible, and especially the extension of the function to the "neuron," the unit of nervous and psychical function considered from every point of view.

Second. All nervous mechanisms of the bulbo-spinal type are organised on a common fundamental basis, and consist of at least two nerve elements—a sensitive epithelial bipolar cell, and a central motor element. Over and above these an intermediate system of cells may be developed, subserving a great variety of functions in all vertebrates.

Third. These intermediate cells of the bulbo-spinal axis allow of co-ordination and correlation of activities between the various metameric segments of the bulbo-spinal tube, and between the several bulbo-spinal centres on the one hand and the encephalic organs on the other.

Fourth. There is an actual and demonstrable basis of structure in the bulbo-spinal system which allows of a great variety in so-called reflex movements; some of these latter being sufficiently elaborate to almost deserve the name "purposive."

Fifth. The cerebral system is a growth and elaboration of neurons in connection with three great sensory projection systems, viz., the *olfactory*, the *optic*, and the *bulbo-spinal* (this last including within itself *tactile, gustatory, kinæsthetic* and other sensory projection systems).

Sixth. The cerebral system has developed at that part of the organism which led in locomotion, was the first to come into contact with the environment, and whose sensitiveness and reactiveness would therefore be the most highly developed in the organism. It is a region, therefore, towards which not only ganglionic concentration takes place; but in which the higher and more complex nerve mechanisms continue in a growing and therefore more plastic state; a highly educable nerve organisation.

Seventh. The cortical mechanism or pallium rises in the scale of mass (quantity) and internal elaboration (quality) as we pass from the amphibian, *viâ* the reptilian, to the mammalian series, finally attaining its highest quantitative and qualitative evolution in man. It becomes the seat and organ of every form and quality of psychical life.

Eighth. The elements which enter into the cerebral organisation in man are the ultimate product of a long series of evolutions in structure and function through countless ages past. These elements are mainly of three kinds, *nervous*, *protective*, and *nutritive.*

Ninth. The cerebral nervous elements (neurons) are separate and distinct anatomically; they do not anastomose one with another, but function only by contact. The individual neuron is a three-fold mechanism, its protoplasmic and nervous processes constituting the two parts which enter into dynamical relationship with other neurons, while the cell protoplasm and nucleus constitute the highly-evolved and specialised trophic and nutritive focus of the whole.

Tenth. The cerebral cortex in man is composed of various cell elements disposed in layers: of these there are four main systems, viz.: the molecular, ambiguous, long pyramidal, and polymorphic, each having its special anatomico-physiological characters.

Eleventh. The sensory excitations passing upwards to the cortex by projection fibres, mainly spread and get distributed in the molecular and sub-molecular regions, affecting thereby both ambiguous cells and long pyramidal cells. This is true of the olfactory and optic projections, and probably true also of the fillet radiations.

Twelfth. While the main fibres and branches of such sensory projection systems are medullated and insulated, the insulation ceases in the "field of conjunction" or nervo-protoplasmic plexuses of the *molecular* and sub-*ambiguous* regions. Here the "naked" collaterals and terminals of these fibres come into the most intimate contact with the enormous expanse of protoplasmic branches and twigs which ramify in those plexus regions.

Thirteenth. A sub-pyramidal nervo-protoplasmic plexus is also formed in the cortex to further increase and perfect the various intrinsic association and commissural systems in the brain.

Fourteenth. That as we rise in the vertebrate scale to the mammalia, a new sub-cortical system of elements (polymorphic cells) is developed. This is even in early life still undergoing demonstrable evolution. It is to be looked upon, therefore, as the latest portion of the cerebral organisation, and one distinctive of the mammalian brain.

Fifteenth. That the whole system of association and commissural fibres grows in accordance with the degree of brain evolution, and that these, together with the polymorphic cell elements, attain their highest relative and absolute evolution in the human brain.

Sixteenth. That the specific natures of psychical processes depend upon the specific activities in the various sensorial "areas" of the cortex, and that the latter are to a certain extent localisable. That the whole body of anatomico-physiological and pathological evidence demonstrates such localisation of function; and that the Golgi method enables us to push this localisation further at least as regards the kinds of cortical nerve elements which are the first to subserve sensorial activities.

Seventeenth. That in disease such centres (according to the locality of the disease) may be the seat of visual, auditory, gustatory, tactile and cutaneous, olfactory or kinæsthetic discharges (hallucinations and Jacksonian epilepsies); and that similarly more extensive and higher cortical areas may be involved, giving rise to "psychical epilepsies."

Eighteenth. That recent evidence and facts tend to show that the Rolandic area is the seat of sensory processes mainly belonging to the kinæsthetic order, concerned, therefore, with the executive activities (bodily *acts*) of the organism in relation to or in adjustment to the environment; and that the whole Rolandic area thus is the finally disposed executive mechanism as far as the brain is concerned.

Nineteenth. That the study of the *aphasias* show that according as various cortical centres and tracts are the locus of the lesion, the clinico-psychological symptoms vary. The whole body of evidence so far considered in our paper, shows that the cerebral cortex is the seat of various psychological activities not only differing in kind, but different also in the form and extent of their elaboration.

Twentieth. That in the Rolandic (kinæsthetic) area a structural evolution is at the root of, and accompanies the functional evolution of movements and acts, the progress of the nerve organisation being parallel to the progress of movements from dyskinæsis to eukinæsis. A similar anatomico-physiological evolution of the cortex takes place in other areas.

Twenty-first. That according to the nature of this anatomico-physiological growth of the brain, it is capable of receiving new impressions and is within certain limits plastic and educable, not a rigidly pre-established structure.

Twenty-second. That quality (extent and complexity) of organisation in the brain is the real basis of intellectual (mental) capacity, and not mere quantity or mass, that thus a brain small in size (like that of Gambetta) may from its intrinsic high elaboration be able to subserve more varied, extensive and multiform activities in life and thought than others of grosser organisation, even if blessed with greater size, can be capable of.

Twenty-third. That the law of psychogenesis takes place in the child not only in the psycho-motor and sensuous, but also in the intellectual (relational) sphere which combines these two, following very definite lines, and that during such stages the child is peculiarly impressionable to surroundings.

Twenty-fourth. That the evolution of language as an instrument for the reinforcement of psychical activities (which, however, can take place in its absence), co-operates greatly in brain development. It allows the child to represent the most varied and different things in terms of a common denominator; it is thus of the highest *formal* value in the exercises and repetitions of psychical processes which underlie mental evolution.

Twenty-fifth. The gradual recognition of the empirical ego as the centre and source of spontaneous activities and its distinction from and antagonism to the external world, are the bases of self-consciousness, self-assertiveness and volition; the higher states of self-sacrifice and altruism arising later (during and after adolescence) in human character.

Twenty-sixth. That under the law of pathogenesis many of these early evolutions in brain and mind, from the fœtal to the adolescent stage, may undergo *alteration* (perversion) or *defect* (absence), and that such pathological conditions of the organism are the basis of the insanities.

Twenty-seventh. That besides lesions in the nerve elements proper, lesions may occur either in the special *protective* or the *nutritive* mechanisms of the nervous system, which we have fully described before, furnishing additional bases towards pathogenesis.

Twenty-eighth. That the whole course of clinico-psychological and pathological evolution in the early stages of an insanity of slow development like alcoholic insanity, enables us to recognise two great classes or types in one of which the morbid changes are more *generalised and extensive,* while in others this first stage is slight or abbreviated, and a *specialised and intensive* development of pathological changes takes place.

Twenty-ninth. That it is possible to correlate the early pathological (internal) changes taking place in the brain with the early clinico-psychological symptoms of the alcoholic insanities, viz.: amnesia, diminished power of attention and volition, diminished initiativeness and energy in conduct, muscular weakness and tremor, and insomnia, and that the further progress of the morbid changes in the brain in association with the psychical conditions of melancholia, with delusions of suspicion, acute hallucinations, maniacal and epileptiform outbursts, are of a more protean but also more advanced type, in which serious destructions (disorganisations) occur chiefly in the nervo-protoplasmic plexuses of the molecular and ambiguous regions on the one hand and in the polymorphic elements on the other; in these cases the nutritive changes *within* the cell-protoplasm and nucleus being also advanced beyond the limits of repair, the changes in the non-nervous elements being also far advanced.

Thirtieth. That the whole of the above body of neurological and pathological evidence, together with recent experimental work, show that in the early stages of the insanities there is a profound *nutritive and dynamical* failure in the nerve elements of the brain which finds expression in the insomnias, the melancholias, and the commencing loss of memory with easily induced mental fatigue which their subjects experience, and that the pathological facts ascertained, in so far as they afford us any light, force on us the conviction that we are dealing with serious nutritive and dynamical changes in the central nervous organ, a conviction which must profoundly influence the clinical study and practical treatment of insanity in the future more than it has in the past.

OBSERVATIONS UPON THE CENTRAL RELATIONS OF THE VAGO-GLOSSO-PHARYNGEAL, VAGO-ACCESSORY AND HYPOGLOSSAL NERVES, FROM THE STUDY OF A CASE OF BULBAR PARALYSIS.

BY WILLIAM ALDREN TURNER, M.D.EDIN., M.R.C.P.LOND.
AND
WILLIAM BULLOCH, M.D.ABERD.

UNDER the terms "progressive muscular atrophy," "progressive bulbar paralysis" and "amyotrophic lateral sclerosis," are described conditions which have pathologically the same morbid substratum, the part affected being the great motor cortico-muscular path in one or other, or both, of its segments.

As regards the upward limits of the degeneration of the pyramidal tracts there is a great variety of opinion, but there can be little doubt that it varies in accordance with the duration of the disease. Kahler and Pick,[1] Charcot and Marie,[2] Lumbroso,[3] and others have been able to trace the degeneration throughout the entire pyramidal tract to the central convolutions of the cortex. Muratoff[4] found degeneration in the internal capsule in a case which he examined. In the cases recorded by Rovighi and Melotti[5] and Dornbluth,[6] the degeneration ceased at the *crura cerebri*. Marie,[7] Kojewnikoff,[8] Joffroy and Achard,[9] and Tooth and

---

[1] Kahler and Pick, *Prager Vierteljahreschrift*, 1879, v.
[2] Charcot and Marie, *Archiv de Neurologie*, 1885, p. 1.
[3] Lumbroso, *Lo Sperimentale*, 1888, lxi., p. 459.
[4] Muratoff, W., *Neurol. Centralbl.*, 1891, p. 518.
[5] Rovighi and Melotti, *Rivista sperim. di Freniatria*, 1888, xiv., p. 315.
[6] Dornbluth, *Neurol. Centralbl.*, 1889, p. 877.
[7] Marie, *Neurol. Centralbl.*, 1884, p. 61.
[8] Kojewnikoff, *Archiv de Neurologie*, 1883, p. 857.
[9] Joffroy and Achard, *Archiv de Méd. et d'Anat. Path.*, 1890, ii., p. 434.

Turner[1] could not trace it beyond the *pons Varolii*, while in Moeli's[2] case it ceased at the pyramidal decussation.

In the spinal cord the degeneration is as a rule limited to the direct and crossed pyramidal tracts, but there may be an extension to the antero-lateral ground bundles, as in the case of Muratoff,[3] who also found degeneration of the posterior longitudinal bundles. Degeneration of the antero-lateral ground bundles has also been found by Charcot and Marie,[4] Leyden,[4] Gombault,[4] Moeli,[4] Marie,[4] Strümpell,[4] and Kronthal.[5] In Muratoff's cases there was also slight degeneration of the mesial fillet and the fibres of the raphé. Strümpell[6] has recorded a case in which the pyramidal tracts entirely escaped, but this is quite an exception.

The bulbar nuclei which are affected also seem to vary, but the hypoglossal nucleus is always more or less atrophied; and next in frequency the facial nucleus. The condition of the *nucleus ambiguus* has been variously stated. Kronthal and Oppenheim[7] found it degenerated in some cases, normal in others. In Muratoff's case it was healthy. The dorsal vagus nucleus has been found degenerated (Muratoff, Kronthal, Lannois and Lemoine[8]), as also the glosso-pharyngeal nucleus (Kronthal). In the cases recorded by Oppenheim, and Tooth and Turner, the dorsal vago-glosso-pharyngeal nucleus was normal.

In cases where the *nucleus facialis* was atrophied the issuing facial roots were only partially degenerated (Muratoff, Tooth and Turner, and Oppenheim).

The motor nucleus of the trigeminus has been found partially atrophied in some cases.

The small-celled hypoglossal nucleus of Roller, and the nucleus of Duval, were normal in the cases of Muratoff, Oppenheim, and Tooth and Turner.

---

[1] Tooth and Turner, BRAIN, 1891, p. 473.
[2] Moeli, *Archiv f. Psych.*, x., p. 718.
[3] Muratoff, *loc. supra cit.*
[4] Quoted by Muratoff.
[5] Kronthal, *Neurol. Centralbl.*, 1891, x., p. 133.
[6] Strümpell, *Neurol. Centralbl.*, 1888, p. 139.
[7] Oppenheim, *Archiv f. Psych.*, 1892, xxiv., p. 758.
[8] Lannois and Lemoine, *Archiv de Méd. Exp. et d'Anat. Path.*, 1894, p. 448.

The oculo-motor nuclei usually escape entirely, but have been found atrophied in some cases.[1]

The changes in the spinal cord chiefly consist of more or less extensive atrophy of the cells of the anterior horns, associated with degeneration in the lateral columns.

Clinical Summary.

Admitted with paresis of the lips, tongue, and soft palate; defect of articulation and swallowing; later on, paresis of vocal cords and atrophy of interosseous muscles of the hands—no affection of the eye muscles or the muscles of mastication. Duration, about two and a-half years. Degeneration of the ganglion cells of the nucleus hypoglossus, nucleus ambiguus, nucleus facialis, *and anterior horns of spinal cord in the cervical and dorsal regions. Slight sclerosis of the crossed pyramidal tracts.*

Lucy V.,[2] age 66, was admitted into King's College Hospital on September 16, 1892, under the care of Dr. Ferrier. The patient, a widow, was by occupation a machine worker. Her habits were temperate. She is stated to have "caught cold" twelve months before admission, from which time she noticed that her speech became thick and indistinct, and that she had difficulty of swallowing. At the same time she suffered from shooting pains in the head.

On admission the patient was in an emaciated anæmic condition. Articulation was hesitating and defective, and there was a nasal tone in her voice. The tongue was puckered at the sides, and tremulous on protrusion. The soft palate rose feebly on phonation and not at all on inspiration, and its reflex irritability was much diminished, as was also its faradic excitability. She was unable to whistle or to pout the lips.

Laryngoscopic examination showed that the vocal cords moved normally. There was difficulty of swallowing, but no affection of the muscles of mastication. The upper and lower limbs, though emaciated, presented no paralytic symptoms, muscular power being good. Both knee-jerks were active, but there was no ankle clonus. The patient remained four months in the hospital, during which time the disease progressed. The movements of the lips and tongue became more impaired, and the voice assumed a more distinctly nasal character, "grub" being pronounced "grum," "rub" being "rum," &c. The interosseous muscles of the hands showed commencing atrophy. There was also wasting of supra- and infra-spinous muscles.

---

[1] Remak, *Archiv f. Psych.*, 1892, xxiii., p. 919.

[2] We are indebted to Dr. Ferrier for the opportunities of studying this case histologically, having had placed at our disposal the resources of the Neuro-Pathological Laboratory, King's College. The clinical notes were from time to time taken by the house physicians, Messrs. Whitfield, Warner and Smith.

She was re-admitted into the hospital in March, 1893, and in addition to the already observed symptoms, weakness of the facial muscles was also noticed, more especially on the right side. The tongue could only be protruded a short distance, and deviated slightly to the right. She was quite unable to pronounce explosive consonants.

The movements of the eyeballs were normal, and the pupils were equal and reacted to light. A laryngoscopic examination showed paralysis of the internal tensors of the vocal cords, so that they did not approximate on phonation. The action of the diaphragm was feeble.

She was discharged in July, 1893. She then passed from observation, and no further notes of her condition were obtained before her death in February, 1894.

PATHOLOGICAL EXAMINATION OF BRAIN AND SPINAL CORD.

The brain and spinal cord were removed, carefully hardened in Muller's fluid and embedded in celloidin, the sections being cut in series and stained by the Weigert-Pal hæmatoxylin process; carmine was used as a counter-stain. Carmine was also used alone, but we found the best ganglion cell stains to be anilin blue and eosin, or nigrosin, in watery solution.

In the *medulla oblongata* and *pons Varolii* the chief pathological changes were found, and mainly in the ganglionic nuclei of those parts, the hypoglossal nucleus being most affected.

The Hypoglossal Nucleus.—At its most distal end, above the decussation of the pyramids, the nucleus contained no ganglion cells, although in one or two sections remnants of a few could be seen. The network of medullated fibres inside the nucleus was, however, to a large extent present, although in comparison with normal sections it appeared to be less dense. Further, the hypoglossal root fibres at this level were completely atrophied. A considerable number of fibres could, however, be traced into the nucleus from the reticular formation. Higher up, the central canal being still closed, a similar appearance was observed. The intranuclear network looked almost normal, but was undoubtedly less dense than in normal sections taken at the same level. At the *calamus scriptorius* one or two shrivelled cells could be seen in the nucleus, but no root fibres proceeding from it. The *fibræ propriæ* of Koch seemed, however, to be fairly well developed. At the level of the middle of the inferior olive, where the nucleus attains its greatest dimensions, the atrophy of the nerve cells and the root fibres was still a marked feature, while the intranuclear medullated network had only partially disappeared. The *fibræ propriæ*, after careful examination (comparison being made with normal sections), could not be regarded as markedly degenerated. The dense tract of fibres at the outer side of the dorsal surface of the nucleus—the dorsal longitudinal bundle of Schütz—appeared to be quite normal. A large number of fibres were observed issuing from the raphé after decussating with similar fibres from the opposite side. These fibres coursed on the ventral aspect of the hypoglossal nucleus, part of them entering it, part proceeding towards the region of the dorsal vago-glosso-pharyngeal nucleus. At the level of the cerebral end of the inferior olive, the ganglion cells and root fibres of the hypoglossal nucleus were also degenerated.

Taking the nucleus as a whole, we came to the conclusion that the ganglionic cells and the root fibres were completely degenerated, while the intranuclear medullated network was only partially degenerated, the *fibræ propriæ* being normal or only very slightly affected. Besides the atrophy of the ganglion cells, the nucleus contained an abnormally large number of rounded neuroglia cells, staining deeply with carmine. The blood vessels were dilated, especially in the dorsal part of the nucleus, so as to separate the fibres of the *fibræ propriæ*. This was very marked in some of the sections; several large distended blood vessels were also seen in the interior of the nucleus, but their walls were not thickened.

The small-celled hypoglossal nucleus, which lies on the ventral side of the proximal end of the main nucleus, and which is regarded by Roller as being a source of hypoglossal root fibres, appeared to be perfectly normal. The nucleus of Duval (*noyaux antero-laterals ou accéssoires* of the hypoglossal) likewise appeared quite normal, when carefully compared with normal sections.

Nucleus Ambiguus.—At the level of the decussation of the fillet no cells could be seen in this nucleus, although in normal specimens individual sections contained three to four cells. From this point up to the *calamus scriptorius* the nucleus was evidently completely degenerated. But, at the *calamus*, sections showed one or two cells in the nucleus, though they were smaller than normal, and their processes were indistinct, clearly being atrophied cells. Slightly higher up, where the nucleus lies near the ascending root of the trigeminus, no cells could be seen, although normal sections show twelve to sixteen multipolar cells at the same level. About the level of the middle of the inferior olive, where the nucleus gains a more median position, lying midway between the *fasciculus solitarius* and the dorsal accessory olive, the ganglion cells were also completely atrophied, and this condition remained up to the upper third of the olive where the nucleus ends.

We could nowhere trace root fibres proceeding from the *nucleus ambiguus*, although in normal specimens these were well marked.

After a very careful examination, and comparison with normal sections, we came to the conclusion that the *nucleus ambiguus* was atrophied throughout its entire extent.

Nucleus Facialis.—[The normal facial nucleus at the level of its greatest dimensions is composed of three parts; a small dorsal part with medium-sized multipolar cells, a middle part with large ganglionic cells, and a large ventral part, also with large ganglion cells.]

At the commencement of the *pons Varolii* the nucleus showed a fair number of ganglion cells, some of which were undoubtedly atrophied. Higher up, in one section ten cells were visible; in another seven; and in a third only four, confined to the dorsal part of the nucleus, the middle and ventral parts being quite devoid of cells. In another section ten cells could be seen in the ventral part of the nucleus, while in the dorsal and middle sub-divisions there were no ganglion cells at all. (Normal nuclei at the same level contain thirty to fifty cells in individual sections.) In its proximal part the nucleus appeared quite atrophied.

In some of the sections a considerable number of fibres could be seen proceeding from the nucleus towards the outer side of the sixth nerve nucleus, but in comparison with normal sections the fibres were fewer in number. The part of the root of the facial nerve which is cut transversely in frontal sections (*genu facialis*) appeared to contain about half the normal number of healthy fibres. The rest were degenerated. The issuing facial roots which come off at a right angle from the *genu facialis* were fairly well marked, but in comparison with normal sections they appeared to be reduced in size by at least one half.

A considerable tract of fibres was seen issuing from the region of the posterior longitudinal bundles, decussating in the raphé, and then coursing ventrally to the *genu facialis*, and dorsally to the abducens nucleus, to make its exit along with the issuing facial roots through the *tegmentum pontis*.

The *nucleus facialis*, as a whole, appeared, therefore, to be atrophied, but not to so great an extent as the *nucleus hypoglossus* or the *nucleus ambiguus*.

In regard to the pyramidal tracts, there seemed to be a slight, diffuse degeneration immediately above the decussation, but higher up in the *medulla*, *pons*, and *crura cerebri*, they were apparently quite normal.

Among structures which appeared to be perfectly normal may be mentioned, the dorsal vago-glosso-pharyngeal nucleus, the *fasciculus solitarius*, the respiratory nucleus of Misslawski (Vorderstrangkern of Obersteiner), the nucleus of Roller, the nucleus of Duval, the cells of the raphé lying ventral to the hypoglossal nuclei, the two small oval nuclei (described by Kölliker, p. 232), which lie between the hypoglossal nuclei, the *nucleus funiculi teretis* of Clarke, the superior olive, the *corpus trapezoides,* the *nucleus abducens,* the *nucleus trochlearis,* the *nucleus oculo-motorius,* the motor and so-called sensory nuclei of the trigeminus, the ascending and descending trigeminal roots, the *lemniscus medialis* and *lateralis,* the nuclei of the *formatio reticularis,* the *nucleus centralis inferior,* the *nucleus reticularis tegmenti pontis;* the *nucleus centralis superior* and *the nucleus lateralis medullæ.*

The Dorsal Longitudinal Bundle of Schütz.—This, which lies in the central grey substance, from the floor of the third ventricle to the spinal cord, and which is regarded by Schütz as giving off fibres to all the cranial nerve nuclei, also appeared to us to be quite normal.

The Spinal Cord.—Examination showed that many of the ganglion cells of the *cervical region* had disappeared, while others were more or less atrophied, this being especially marked in the ventro-mesial, dorso-mesial, and central groups; of the ventro-lateral and dorso-lateral groups the latter was the less affected, and, indeed, appeared to be normal. Apart from the cells of this group and a few of the ventro-lateral group, no large multipolar ganglion cells, with distinct processes, could be seen. A few cells were present in the *processus reticularis.* Further, sections stained by hæmatoxylin showed that a large part of the network of medullated fibres in the anterior horn had become broken up, and the anterior motor roots were much degenerated, in some sections none being visible at all.

The position of the nucleus of the fibres of the spinal accessory nerve (*accessorius spinalis*) is variously located by authorities, but the ganglionic cell group which we regard as giving origin to this nerve lies in the upper segments of the cord, internal to the *processus reticularis,* and posterior and internal to the postero-lateral group of nerve cells of the anterior horn. No issuing nerve fibres of the root could be detected in any of the sections, and the cells of this nucleus, although not completely atrophied, showed considerable degenerative change. The crossed pyramidal tracts showed a slight, diffuse degeneration. The anterior white commissure could not be said to be materially affected.

In the *dorsal region* most of the ganglion cells of the anterior horn had disappeared. The cells seemed fairly normal in the *cornu lateralis*, in front of which a few cells could also be seen with distinct processes (probably the continuation of the ventro-lateral and dorso-lateral groups of the cervical region). The cells of Clarke's column were normal. The anterior roots were much atrophied, and there was also a marked disappearance of the medullated fibres of the anterior horn itself. The degeneration of the crossed pyramidal tracts was much the same as in the cervical region, but, if anything, more diffuse.

In the *lumbar region* the cells were in very distinct groups, and were evidently not so atrophied as in the cervical and dorsal regions. A considerable number was seen in the ventral groups, and the dorso-lateral group contained many normal multipolar cells. The degeneration in the pyramidal tracts was very slight, and could only be recognised with difficulty.

The posterior nerve roots were normal throughout, as also were the ganglion cells of the posterior horn. Many irregularly-scattered, apparently normal cells were also found at the junction of the anterior and posterior horns. The disease had affected chiefly the cervical and dorsal regions, the lumbar having escaped to a large extent, while in the affected regions, though most of the cell groups of the anterior horn were more or less atrophic, the *dorso-lateral* group was, after careful examination, considered to be normal. The diffuse degeneration above described in the crossed pyramidal tracts seemed to be a part of a slight, diffuse peripheral myelitis, involving the antero-lateral columns. This was so marked in some sections as to resemble degeneration of the fibres of the antero-lateral ascending tract of Gowers.

CRITICAL REVIEW OF THE CONNECTIONS OF THE AFFECTED NUCLEI.

(1) *The Hypoglossal Nucleus.*—As already pointed out, the cellular element is that which has undergone the greatest amount of degeneration; on the other hand, the *fibræ propriæ,* and the fibres which pass from the raphé into and around the nucleus, retain a normal appearance.

The true nature of the *fibræ propriæ* has been the subject of much discussion. They were regarded by Koch,[1] who originally described them, as fibres of a system which connected individual cells of the hypoglossal nucleus at different levels, some of them being also commissural fibres between the two nuclei. Obersteiner[2] regards them as the fibres of the dorsal longitudinal bundle, which is here better developed than elsewhere.

We desire to state the following observations, in regard to the *fibræ propriæ,* from a careful examination of fœtal and pathological conditions.

(*a*) In a case of degeneration of both pyramidal tracts associated with complete atrophy of the hypoglossal nucleus and its root fibres,[3] the *fibræ propriæ* had completely disappeared, with the exception of a small area in the dorso-lateral part of the nucleus, between the dorsal vago-glosso-pharyngeal nucleus and the *nucleus funiculi teretis.* This small area occupies the position of the dorsal longitudinal bundle of Schutz, as figured by him.[4] As in this case (Tooth and Turner) the raphé fibres on the ventral aspect of the nucleus were normal, they cannot be regarded as coming from the pyramids.

(*b*) In the case under discussion, in which the ganglion cells of the hypoglossal nucleus were atrophied while the pyramidal tracts were almost unaffected, a considerable part of the intranuclear network was still present, and the *fibræ propriæ* seemed to be as well developed as in normal sections. The dorsal longitudinal bundle and the ventral raphé fibres were also healthy.

---

[1] Koch, *Archiv f. Mikroskop. Anat.*, 1888, p. 54.

[2] Obersteiner, "Nervosen Centralorgane," second edit., 1892, p. 399.

[3] Tooth and Turner, BRAIN, 1891, p. 473.

[4] Schütz, *Archiv f. Psych.*, 1891, p. 527.

(*c*) In an eight months human fœtus, where the pyramidal fibres had not received their medullary sheaths, the *fibræ propriæ* were also non-medullated, the only medullated fibres in the nucleus being the pencil-like arrangement of the root fibres. In a full time fœtus, where the pyramids had begun to medullate, there could be seen a faint indication of *fibræ propriæ* on both sides, while the raphé fibres on the ventral aspect of the nucleus were fully medullated.

(*d*) In a case of unilateral sclerosis of the pyramid with degeneration of the mesial fillet, the difference between the *fibræ propriæ* on the two sides was, however, inappreciable; although after careful examination we came to the conclusion that they were less developed and less dense than normal. In this case there was no difference between the bundles of Schütz and the ventral fibres from the raphé.

(*e*) In a series of sections from the *medulla oblongata* of a kitten, stained by the rapid osmium-silver method, we were able to trace long fibres from the nucleus into and through the raphé towards the pyramids.

We would, therefore, deduce from these observations: firstly, that the hypoglossal segment of the bundle of Schütz does not undergo atrophy in cases of bulbar paralysis, either when the ganglionic cells of the hypoglossal nucleus are atrophied alone, or in conjunction with sclerosis of the pyramidal tracts; secondly, that the fibres which pass from the raphé ventral to the nucleus are not pyramidal fibres; and, lastly, that the *fibræ propriæ* themselves are in all probability chiefly pyramidal fibres.[1]

Kölliker[2] describes a number of fibres entering the hypoglossal nucleus from the *substantia reticularis grisea* between the hypoglossal and vago-glosso-pharyngeal roots. These fibres were present in large numbers in our case, and were also seen in that of Tooth and Turner. The probability, therefore, is that they are not pyramidal fibres, for they were also present in our embryonic series when the pyramids were not medullated. It is not improbable, as Kölliker (*loc. cit.*, p. 235) suggests, that they may be derived from some of the longitudinal fibres of the *formatio reticularis*. So far, we have been unable to trace them to cells in this formation. Fibres undoubtedly pass from the posterior longitudinal bundles to the hypoglossal nuclei; and commissural fibres between the two hypoglossal nuclei also exist, and form a well-marked system.

---

[1] See also Mingazzini, *Annali di Freniatria*, 1891.

[2] Kolliker, "Handbuch der Gewebelehre," 1894, p. 284.

From these considerations we therefore suggest the following connections and relations of the hypoglossal nucleus:—

(*a*) There are *efferent* and *afferent* fibres in connection with the hypoglossal nucleus, the efferent being the root fibres.

(*β*) Fibres pass from the raphé in a ventro-dorsal direction to gain the dorsal surface of the nucleus, where they, passing between the two nuclei, appear to run in a longitudinal direction, ultimately forming a large part of the *fibræ propriæ* of the nucleus.

(*γ*) Another set from the raphé courses on the ventral surface of the nucleus (*fibræ afferentes* of Koch). Of these some enter the nucleus, while others pass towards the vagus roots and vagus nucleus. Their origin is uncertain. Most of them do not seem to course for any long distance in the raphé, but cross it shortly after entering.

(*δ*) A set of fibres passes obliquely through the *formatio reticularis grisea* into the nucleus, chiefly marked at its caudal extremity.

(*e*) Fibres enter the nucleus from the posterior longitudinal bundles.

(*ζ*) Commissural fibres exist between the two nuclei.

(*η*) The small-celled nucleus of Roller and the nucleus of Duval do not give origin to hypoglossal fibres as described by those authors.

(*θ*) The cells of the raphé ventral to the hypoglossal nucleus do not give origin to root fibres.

(*ι*) The classical hypoglossal nucleus of Stilling is the main, if not the only, source of the hypoglossal nerve roots.

(2) *The Nucleus Ambiguus* (or ventral vago-glosso-pharyn-

geal nucleus).—The most complete description of this difficult nucleus has been given by Kölliker (*loc. cit.*, p. 241). Our own observations in this case and in others lead us to make the following remarks. The nucleus varies somewhat in position, according as it is viewed in sections from the lower or upper regions of the *medulla oblongata*. In the distal parts of the *medulla* it lies more external, and near the *substantia gelatinosa Rolandi*, and it has here to be carefully distinguished from the antero-lateral nucleus which lies quite close to it on its outer side. The latter nucleus is distinguished mainly by the large amount of neuroglia among which its cells lie. In the proximal parts it lies between the *fasciculus solitarius* and the dorsal accessory olive. Here it has to be distinguished from the *nucleus centralis inferior*, which lies on its mesial side. The *nucleus ambiguus* is co-extensive with the dorsal vago-glosso-pharyngeal cell column, and according to Kölliker it is the motor nucleus of the vago-accessory and vago-glosso-pharyngeal nerves. The *accessorius vagi* is a pure motor nerve, while the vago-glosso-pharyngeal nerves are mixed, *i.e.*, they contain both motor and sensory fibres (Kölliker). The cells of the *nucleus ambiguus* are large and multipolar. They often appear elongated, the long axis of the cell lying in the direction of the fibres issuing from the nucleus. Vincenzi[1] has also figured a cell of Golgi's second type in it, but in our sections from the brains of kittens, we have been able to find only first type cells.

The condition of the nucleus in cases of bulbar paralysis has been variously stated by observers. Of four cases carefully examined by Oppenheim,[2] the nucleus was healthy in two; in the two in which it was degenerated, there had been observed during life paralysis of the internal thyro-arytenoid muscles. This association was also present in our case. Taking the well-known clinical combination of paralysis of the soft palate and of the vocal cords (Hughlings-Jackson) in conjunction with the physiological experiments upon the vagus and vago-accessory roots (John Reid, Cl. Bernard,

---

[1] Vincenzi, ref. in Kölliker, *loc. cit.*

[2] Oppenheim, *Archiv f. Psych.*, 1892, p. 758.

Beevor and Horsley), and the pathological facts recorded by Oppenheim and ourselves, we would argue that *the nucleus ambiguus is the motor nucleus for the elevators of the soft palate and the tensors of the vocal cords.*

The lower portion of the dorsal vago-glosso-pharyngeal nucleus, which has up till recently been regarded as the nucleus of origin of the vago-accessory fibres, was normal in our case.[1] From the fact that the vago-accessory root fibres were completely atrophied, while the vago-glosso-pharyngeal root fibres were only partially degenerated, we conclude that the former is a purely motor nerve arising in the *nucleus ambiguus*, while the latter contains both efferent (motor) and afferent fibres.

(3) *The Dorsal Vago-glosso-pharyngeal Nucleus.*—This nucleus is one long cell column, lying at first where the central canal is still closed, dorsal to the hypoglossal nucleus. Higher up it lies lateral to the latter, forming the *ala cinerea* in the floor of the fourth ventricle. It has been customary to differentiate the nucleus roughly from above downwards (Stilling,[2] Dees,[3] &c.) into three parts, glosso-pharyngeal, vagus, and accessory. This segmentation appears to us needless, as it is quite impossible, either from the character of the cells or otherwise, to distinguish such groups. Our observations from the case under discussion, taken into consideration with the study of sections of the *medulla oblongata* of newly-born kittens, prepared by the rapid osmium-silver method, confirm the views originally expressed by His,[4] Kölliker[5] and Held[6]—that the whole of this cell column is the end nucleus ("endkern") of the vago-glosso-pharyngeal nerve roots. The ganglion cells are seldom affected in bulbar paralysis. There are two kinds of cells in the nucleus, as Held has pointed out; this our observations in the kitten confirm. In one kind the axis-cylinder does

---

[1] This nucleus was also unaffected in the case of Tooth and Turner, *op. cit.*, p. 473.

[2] Stilling, "Ueber die Medulla Oblongata," p. 24, 1843.

[3] Dees, *Archiv f. Psych.*, xx., p. 89.

[4] His, *Archiv f. Anat. und Phys. Anat. abth.*, 1887, p. 368.

[5] Kölliker, *Op. cit.*

[6] Held, *Archiv f. Anat. und Physiologie Anat. abth.*, v. und vi., p. 435.

not leave the grey matter of the nucleus, while in the other it can be traced beyond the nucleus for a considerable distance. Some of the axis-cylinders in our specimens could be traced outwards into the lateral part of the *formatio reticularis*, where they were gradually lost, others passing towards the raphé, where they disappeared from view. The axis-cylinders, described by Held as going to the opposite interolivary layer, we were unable to find. There can be little doubt that these axis-cylinder processes of the cells of the nucleus are the sensory paths of the "second order," connecting the vago-glosso-pharyngeal cells with other parts of the central nervous system. The cells from which the afferent vago-glosso-pharyngeal nerve fibres spring are situated in the petrosal and jugular ganglia in the case of the glosso-pharyngeal, and in the ganglia of the root and trunk in the case of the vagus nerve.

(4) *Nucleus Facialis.*—The general features in regard to the connection of this nucleus with its root fibres are fairly well understood. The upper facial division, which supplies the *orbicularis palpebrarum* and *frontalis* muscles, is believed to arise in relation to the oculomotor nucleus. From this the fibres pass by the posterior longitudinal fasciculus, to issue along with the facial roots. Our case confirms the observations of Tooth and Turner (*op. cit.*), Oppenheim (*op. cit.*), and Muratoff,[1] who observed proceeding from the posterior longitudinal bundle, and decussating in the raphé, a set of fibres which passed ventral to the *genu facialis* and dorsal to the abducens nucleus to issue with the facial nerve as already described. Such a connection is, however, denied by Duval and Kolliker.[2]

(5) *The Spinal Cord.*—As regards the spinal cord, our observations permit us to remark that while many of the cell groups, ventro-mesial, dorso-mesial, central and ventro-lateral are largely atrophied, individual healthy cells may yet be seen.

---

[1] Muratoff, *Neurolog. Centralbl.*, 1891, x., p. 514.

[2] Bregmann (*Jahrbuch. fur Psychiatrie*, Bd. xi., Heft 1 and 2), who divided the facial nerve extra-cranially in animals, was unable to trace degeneration in the posterior longitudinal bundles, although the root fibres and nucleus were affected.

These latter are not true motor cells, but belong to the now well-recognised system of "column cells" (Strangzellen). We would especially refer to the apparently normal condition of the dorso-lateral cell group throughout the cord. When viewed with reference to the condition of the cells of the anterior cornua, the case under discussion may be regarded as comparatively recent. We have had, however, the opportunity of comparing our sections with those of a case of old-standing amyotrophic lateral sclerosis. In this latter, the cells of all the groups in the anterior horns have disappeared, with the exception of those of the dorso-lateral group, which is, however, partially atrophied.

These observations harmonise with the views of Kölliker and Mingazzini[1] that the dorso-lateral cell-group of the anterior horn, though giving axis cylinder processes to the anterior nerve roots, stands in relation much more to the collaterals of the posterior radicular fibres than to those of the pyramidal tracts. From this, they argue that the cell group is closely associated with the reflex mechanisms. We suggest that this probably explains the retention of the knee-jerk in cases of advanced progressive muscular atrophy.

GENERAL SUMMARY.

(1) The hypoglossal chief nucleus (Stilling) is the only source of origin of the root fibres of the hypoglossal nerve. The *fibræ propriæ* of the nucleus are in large part derived from the pyramids. The other afferent fibres come from the *formatio reticularis.*

(2) The *nucleus ambiguus* is the nucleus of origin of the motor vago-glosso-pharyngeal and vago-accessory root fibres, and is the source of innervation of the *levator palati*, and internal thyro-arytenoid muscles, and probably also of the muscles of the pharynx.

(3) The posterior vago-glosso-pharyngeal nucleus is the end nucleus of the axis-cylinder processes of the cells in the

---

[1] Mingazzini, *Rivista Sperim. di Freniatria*, 1892, p. 472.

ganglia upon the trunk of the glosso-pharyngeal and vagus nerves.

(4) The facial nerve trunk receives a large accession of fibres from the posterior longitudinal bundle of the opposite side, which probably comes from the oculomotor nucleus.

(5) The postero-lateral cell group of the anterior horn of the spinal cord is least and latest affected in chronic progressive poliomyelitis; and, as shown by Mingazzini, is probably associated with the reflex mechanisms of the spinal cord.

Abstract.

Henschen on the Anatomy of the Visual Path.

THE visual path from the bulb of the eye to the visual centre in the calcanean fissure of the occipital lobe consists anatomically of knots, besides those which may be present in the retina and in the cerebral cortex. The anterior portion of this knot is made up of the great ganglion cells of the retina and of the nerve-fibres, which proceed backward and form the optic nerve, the chiasma, and the tractus, and ramify into the corpora geniculata. The posterior portion consists of the ganglion cells in the corpora geniculata and the nerves which extend from the occipital visual path into the cortex of the visual centre. The visual nerves consist of crossed and uncrossed and macular fibres. The macular fascicle, which in the papilla is situated latero-ventrally, afterwards reaches the centre, taking its course into the chiasma and the tractus as a central fascicle. The uncrossed fibres are situated in the papilla, stratified into two separate fascicles, one latero-dorsal, the other latero-central, separated by the macular fascicle. Farther back in the optic nerve these separate fascicles reunite to form a lateral. The crossed fascicle is situated in the papilla, medially or dorso-medially. In the chiasma both the fascicles are intertwined, the crossed fascicle later on lying ventro-medially and the uncrossed latero-dorsally in the tractus. The fibres which correspond to the dorsal half of the retina are situated dorsally in both of the fascicles, these fascicles being separated until they reach the corpus geniculatum externum, where their filaments lie close together. The filaments of the optic nerve partly form the capsule of the ganglion and partly radiate into the ganglion and form the principal part of the so-called medullar lamella. The geniculate ganglion is also the chief visual ganglion, but filaments from the optic nerve enter as far as the pulvinar and the corpora quadrigemina anteriora, and any defect in the field of sight does not seem to arise from destruction of these fibres. Filaments from the cells of the geniculate ganglion extend into the so-called occipital visual path, forming a fascicle, about five millimetres in thickness, on the level of the first temporal groove and the second temporal convolution, running backward toward the bottom of the fissura calcarina. In this visual path are the filaments for the dorsal quadrant of the retina, situated dorsally just as in the frontal visual path. From this fascicle run out into the occipital lobe fascicules of the filaments, curving toward the bottom of the fissura calcarina and its labia, where the optic centre is situated. The visual centre is essentially limited to the cortex of the fissura calcarina, and a unilateral destruction of this cortex produces complete hemianopsia. The upper portion of this centre represents the superior and the lower the inferior half of the retina. The macular portion is probably situated in the bottom of the fissura calcarina, more toward the front, and the peripheral retinal field more occipitally. The macular field of the retina is often bilaterally innervated from both halves of the brain. The elements of the crossed and uncrossed fibres do not occupy microscopically separate fields in the cortex, but are situated quite close to each other. In this so-called visual centre arise the perception of light as well as of colour. Besides filaments uniting the bulb with the anterior corpora quadrigemina and the corpora pulvina, there exist also filaments uniting the visual centre with the ganglia, probably reflex filaments. The theory of the French school, that the visual path participates in the formation of the internal capsules or that it crosses once (Charcot) or several times (Grasset) behind the chiasma, is regarded by the author as erroneous.—*Abst. in Univ. Med. Journal*, 1894.

Rules

AND

LIST OF OFFICE BEARERS AND MEMBERS

OF THE

Neurological Society of London,

1894,

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1893.

LONDON
JOHN BALE & SONS
87-89, GREAT TITCHFIELD STREET, OXFORD STREET, W.
1894.

Office Bearers for the Year 1894.

President.

DAVID FERRIER, M.D., F.R.S.

Vice-Presidents.

SIR WILLIAM H. BROADBENT, BART., M.D., F.R.C.P.
D. HACK TUKE, M.D., LL.D., F.R.C.P.

Council.

HENRY CHARLTON BASTIAN, M.D., F.R.S.
J. S. BRISTOWE, M.D., F.R.S.
THOMAS BUZZARD, M.D., F.R.C.P.
BYROM BRAMWELL, M.D., F.R.C.P.E.
ALEX. HILL, M.A., M.D.
ROBERT MAGUIRE, M.D., F.R.C.P.
WILLIAM MILLER ORD, M.D., F.R.C.P.
C. S. SHERRINGTON, M.D., F.R.S.
AUGUSTUS WALLER, M.D., F.R.S.
A. DE WATTEVILLE, M.D., B.Sc.

Treasurer.

CHARLES EDWARD BEEVOR, M.D., F.R.C.P.

Secretaries.

H. H. TOOTH, M.D., F.R.C.P.
F. W. MOTT, M.D., F.R.C.P.

Neurological Society of London.

REPORT OF COUNCIL.

JANUARY 25th, 1894.

The Council begs to report to the Society its continued prosperity. The total number of Members is now 152, and the interest shown in the work of the Society has been even more evident during the past year than in previous sessions. It has been found necessary to hold more than the usual number of meetings, and the average attendance of Members has increased.

At the Annual General Meeting, held on January 19th, 1893, the newly-elected President, Professor SCHÄFER, delivered an address on "The Nerve Cell as the basis of Neurology"; and a full list of the communications to the Society during the year is appended to this Report.

The Balance in the hands of the Treasurer is again satisfactory, as will be seen from the accompanying Balance Sheet; and "Brain," the Journal of the Society, edited by Dr. DE WATTEVILLE, has maintained its high reputation.

But while reporting that the condition of the Society is in so many respects satisfactory, the Council regrets the loss to the Society of two greatly esteemed Members. Professor CHARCOT, lately elected an Honorary Member of the Society, died during the year, and the Science of Neurology has lost in him one of its most able exponents. The death of Dr. JAMES ANDERSON affected the Society even more nearly. Dr. ANDERSON was for several years Secretary to the Society, and at the time of his death was a member of the Council. The Society was greatly indebted to him for much of its success, and his work was highly valued.

List of Communications to the Neurological Society, 1893.

January 10th, Professor SCHÄFER, F.R.S. Inaugural address, "The Nerve Cell as the basis of Neurology," *Brain*.

February 9th, Dr. SHERRINGTON, F.R.S. "The Skin Fields of some Sensory Spinal Nerve Roots."

May 11th, Dr. RISIEN RUSSELL. "On the results of Cerebellar Lesions in Dogs." Professor HORSLEY, F.R.S., and Dr. KRAMER, "On an Experimental Investigation of the Effects of Gun Shot Wounds of the Brain." Dr. MOTT, "The Effects of Removal of Cortical Motor Centres." Dr. HALE WHITE, "The Effects of Removal of 'the Arm Area' of the Cortex." Dr. SHERRINGTON, F.R.S., "Division of the Fifth Nerve."

May 15th, Mr. R. BOYCE, Photographs illustrating the degeneration in the Medulla and Cord following ablation of the Cerebral Hemispheres in Mammals. Dr. SHERRINGTON, F.R.S., Photographs illustrating the Skin Fields of the Upper Sensory Roots. Drs. BRUNTON, F.R.S., and CARRUTHERS, "Experiments in Elucidation of 'Treadler's Cramp.'" Dr. A. S. GRUNBAUM, "On the Local Fibres of the Spinal Segment." Professor SCHÄFER, F.R.S., and Dr. FLOOD, "Some Results of Extirpation of the Lower Part of the Ascending Frontal Gyrus."

June 15th, Dr. J. HUGHLINGS JACKSON, F.R.S., "Ophthalmoplegia Externa, with weakness of the Orbicularis Palpebrarum." Dr. FERRIER, F.R.S., "Ophthalmoplegia Externa, with weakness of the Orbicularis Palpebrarum." Dr. WOOD, "Tabes with Ophthalmoplegia, Paralysis of the Motor part of fifth nerve, and Laryngeal Paralysis." Dr. J. HUGHLINGS JACKSON, F.R.S., "Lesions of the Sixth, Seventh, and Twelfth Cranial Nuclei, with Paralysis of the Infra-hyoid Muscles." Dr. JUDSON BURY, "Persistent profound Hemianæsthesia, with marked Concentric Narrowing of the Visual Fields, and Trophic Lesions of the Hand on the affected side."

November 23rd, Dr. DONKIN, "A Case of Muscular Wasting of both Upper Extremities, and complete Paralysis of one." Dr. H. LEWIS JONES, "Five Cases of Symmetrical Atrophy of the Intrinsic Muscles of the Hand in Young People." Dr. ORMEROD, "A Case of Syringo-myelia, showing loss of temperature sense, but not of pain." Dr. BEEVOR, "Paralysis Agitans, with Tremor of Lips, Tongue, and Jaw." Dr. HECTOR MACKENZIE, "A Case of Friedreich's Disease; a Case of Chorea in a man aged 81."

BALANCE SHEET, 1893.

NEUROLOGICAL SOCIETY.

Cr. Receipts.	£	s.	d.
To Balance in hand	169	0	10
Subscriptions for current-year	142	0	0
Do. arrears	5	0	0
Do. in advance	1	0	0
Sundries	0	12	0
Plates refunded	4	0	0
Share of profit on "Brain" (Messrs. Macmillan & Co.)	4	17	2
	£326	10	0

Expenditure. *Dr.*	£	s.	d.
By Illustrations—Danielssen	4	0	0
Do. Thevoz & Co.	12	0	0
Do. Griffiths	8	8	0
Do. Typographic Etching Co.	8	11	0
Do. Watkinson & Co.. ...	6	10	0
Stationery, &c.—Pulman & Sons ...	17	11	3
Do. and Reprints—Bale & Sons ...	9	2	11
Sundries (Scotch Drafts)...	0	3	0
Housekeeper, University College ...	3	1	0
Royal Medico-Chirurgical Society—Hire of rooms, £9 9s.; Refreshments, £5 5s.	14	14	0
Messrs. Macmillan—144 Subscriptions of Members at 11s. each, £79 4s.; Do. on additional Subscriptions, £6 12s.; Excess of Corrections on "Brain," £2 ...	87	16	0
Messrs. Coutts' cheque book	0	4	0
Balance in hand, Dec. 13, 1893... ...	154	8	10
	£326	10	0

Audited and found correct,

A. de Watteville.

RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF LONDON.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Pound, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due shall be considered as equivalent to resignation.

15.—Absence of any Member residing within the Metropolitan area from all Meetings held during the year shall be considered as equivalent to resignation.

16.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, and ten Councillors.

17.—Five Members of the Council shall form a quorum.

18.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

19.—The office of Vice-President shall be tenable for two years; one Vice-President being elected every year.

20.—The Secretaries and Treasurer shall be elected annually, with eligibility for re-election.

21.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

22.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

23.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

24.—There shall be six ordinary meetings annually, of which the first held in each year shall be the Annual General Meeting.

25.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

26.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members.

27.—The President shall have the power of inviting any person to attend and to take part in the scientific work of a meeting.

28.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a Member, they shall submit the question to a Special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

29.—The Council shall draw up and submit for the approval of the Society supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

30.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by A. de Watteville, M.A., M.D., B.Sc.

HONORARY MEMBERS.

Professor HELMHOLTZ, Berlin.
Mr. HERBERT SPENCER, London.
Professor WUNDT, Leipzig.

CORRESPONDING MEMBERS.

Professor BROWN-SÉQUARD, Paris.
„ ERB, Leipzig.
„ HITZIG, Halle.
„ JOLLY, BERLIN.
„ SCHIFF, Geneva.
Dr. WEIR MITCHELL, Philadelphia.

⁂ Members are requested to communicate with the Secretaries when corrections are necessary.

EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.
Pres., President.
V.P., Vice-President.
Tr., Treasurer.
Sec., Secretary.
C., Member of Council.

ORDINARY MEMBERS.

Elected.	
1892	**Alexander, Robert Reid**, M.D., The Asylum, Hanwell, W.
1891	**Ballance, C. A.**, M.S., F.R.C.S., 106, Harley Street, W.
O.M.	**Barlow, Thomas**, M.D., F.R.C.P., 10, Wimpole Street, W.
,,	**Bastian, H. Charlton**, M.D., F.R.S., 8a, Manchester Square, W. (Pres., 1892; V.P., 1891; C., 1886-9, 1893.)
,,	**Beach, Fletcher**, M.B., F.R.C.P., The Elms, Chislehurst Road, Sidcup, Kent.
,,	**Beevor, C. E.**, M.D., F.R.C.P., 33, Harley St, W. (Tr., 1894; C. 1893.)
,,	**Bennett, A. Hughes**, M.D., F.R.C.P., 76, Wimpole Street, W. (Sec., 1886-7; C., 1888-91.)
1888	**Birt, Ernest**, L.R.C.P., M.R.C.S., West Riding Asylum, Wakefield.
1892	**Bowlby, Anthony A.**, F.R.C.S., 24, Manchester Square, W.
1893	**Bowman, Henry Moore**, M.D., M.R.C.P., 2, Museum Chambers, Bury Street, W.C.
1889	**Bradford, J. R.**, M.D., D.Sc., M.R.C.P., 52, Upper Berkeley Street, W.
O.M.	**Bramwell, Byrom**, M.D., F.R.C.P.Ed., 23, Drumsheugh Gardens, Edinburgh (C., 1894)
,,	**Bristowe, J. S.**, M.D., F.R.S., 13, Old Burlington Street, W. (Pres., 1891; V.P., 1889-90; Tr., 1886-8; C., 1892-3.)
1892	**Bristowe, Hubert Carpenter**, M.D., L.R.C.P., M.R.C.S., County Asylum, Wells, Somerset.
O.M.	**Broadbent, Sir Wm. H.**, Bart., M.D., F.R.C.P., 84, Brook Street, W. (C., 1886-7; V.P., 1893.)
1888	**Brown, Sanger**, M.D., West 173rd Street, Fort Washington, New York.
O.M.	**Bruce, Alex.**, M.D., F.R.C.P.Ed., 13, Alva Street, Edinburgh.
,,	**Bruce, J. Mitchell**, M.D., F.R.C.P., 70, Harley Street, W.
,,	**Brunton, T. Lauder**, M.D., F.R.S., 10, Stratford Place, W.
1890	**Bury, Judson Sykes**, M.D., M.R.C.P., 10, St. John Street, Manchester.
O.M.	**Buzzard, Thomas**, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.P., 1888-9; C., 1886-7, 1891-3.)
1891	**Cagney, James**, M.D., M.Ch., M.R.C.P., 93, Wimpole Street, W.
1889	**Carter, Robert Brudenell**, F.R.C.S., 27, Queen Anne Street, W.

1887 Cattell, J. McK., M.A., Ph.D., Columbia College, New York, U.S.A.
1891 Clarke, J. Michell, M.B., M.R.C.P., 28, Pembroke Road, Clifton, Bristol.
O.M. Cobbold, C. S. W., M.D., F.R.C.P.Ed., Bailbrook House, Bath.
1892 Cole, Robert Henry, M.B., L.R.C.P., M.R.C.S., Moorcroft, Hillingdon, Uxbridge.
1890 Colman, W. S., M.D., M.R.C.P., 72, Wimpole Street, W.
1890 Corner, Harry, M.D., Bethlem Royal Hospital, Lambeth, S.E.
O.M. Donkin, H. B., M.B., F.R.C.P., 108, Harley Street, W.
" Down, J. Langdon, M.D., F.R.C.P., 81, Harley Street, W.
" Dreschfeld, Julius, M.D., F.R.C.P., 325, Oxford Road, Manchester.
" Drummond, David, M.D., 6, Saville Place, Newcastle-on Tyne.
1892 Dupuy, Eugène, M.D., 53, Avenue Montaigne, Paris.
1889 Eccles, A. Simons, M.B., M.R.C.S , 23, Hertford Street, Mayfair, W.
O.M. Edmunds, Walter, M.D., F.R.C.S., 75, Lambeth Palace Road, S.E.
" Ferrier, David, M.D., F.R.S., 34, Cavendish Square, W. (Pres. 1894; V.P , 1892-3; C., 1886-90.)
" Foster, Michael, M.D., F.R.S., Shelford, Cambridge.
1888 Fox, Arthur, M.B., F.R.C.P.Ed., 16, Gay Street, Bath.
O.M. Fox, E. Long, M.D , F.R.C.P., Church House, Clifton, Gloucestershire.
" Gaskell, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs.
1887 Gay, Wm., M.D., M.R.C.P., Brookland, Hemel Hempstead.
O.M. Gee, Samuel J., M.D., F.R.C.P., 31, Upper Brook Street, W.
" Godlee, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.
1890 Goodall, E. W., M.D., Eastern Hospitals, Homerton, E.
1892 Gotch, Francis, M A., F.R.S , 11, Prince's Park, Liverpool.
O.M. Gould, A. Pearce, M.B., F.R C.S., 10, Queen Anne Street, W.
1893 Grant, Dundas, M.A., M.D., F.R.C.S , 8, Upper Wimpole Street, W.
O.M. Gunn, R. Marcus, M.B., F.R.C.S., 54, Queen Anne Street, W.
1893 Halliburton, William Dobinson, M.D., F.R.S., 9, Ridgemount Gardens, W.C.
O.M. Hamilton, D. J., M.B., F.R.S.E., University, Aberdeen.
1889 Handford, Henry, M.D., M.R.C.P., 14, Regent Street, Nottingham.
1891 Hawkins, Herbert Pennell, M.B., M.R C.P., 109, Harley Street, W.
O.M. Haycraft, J. B., M.B., F.R.S.E., 14, Westbourne Terrace Road, W.
1891 Head, Henry, M.B , C.M., 6, Clarence Terrace, Regent's Park, N.W.
O M. Hill, Alex , M.A., M D., Downing Lodge, Cambridge. (C., 1892.)
" Hopkins, John, F.R.C.S., London Sick Asylum, Cleveland Street, W.
" Horsley, Victor, F.R.C.S., F.R.S., 25, Cavendish Square, W. (C., 1892-3)
1889 Huggard, William R , M.D., M.R.C.P., Davos Platz, Switzerland.
1889 Humphry, Lawrence, M.D., 3, Trinity Street, Cambridge.
O.M. Hutchinson, Jonathan, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres. 1889; V.P., 1887-88; C., 1886, 1890.)

1889 Hyslop, T. B., M.B., C.M., Bethlem Royal Hospital, Lambeth, S.E.

O.M. Jackson, J. Hughlings, M.D., F.R.S., 8, Manchester Square, W. (Pres., 1886; C., 1887-92.)

,, Jessop, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1892 Jones, Henry Lewis, M.D., M.R.C.P., 9, Upper Wimpole Street, W.

O.M. Langley, J. N., M.A., F.R.S., Trinity College, Cambridge.

1888 Lawford, J. B., M.D., F.R.C.S., 55, Queen Anne Street, W.

O.M. Lees, D.B., M.D., F.R.C.P., 22, Weymouth Street, W.

1891 Macdonald, Peter William, M.D., C.M., The County Asylum, Dorchester, Dorset.

1891 Mackenzie, Hector William Gavin, M.D., F.R.C.P., 59, Welbeck Street, W.

O.M. Mackenzie, Stephen, M.D., F.R.C.P., 18, Cavendish Square, W.

1889 Macphail, S. R., M.D., Borough Asylum, Rowditch, Derby.

O.M. Maguire, Robert, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C. 1893.)

,, Mann, J. Dixon, M.D., F.R.C.P., 16, St. John Street, Manchester.

1893 Maude, Arthur, L.R.C.P., M.R.C.S., Westerham, Kent.

1891 May, W. Page, M.D , M.R.C.S., National Hospital, Queen's Square, W.C.

1890 Menzies, William Francis, M.D., B.Sc., M.R.C.P., County Asylum, Rainhill, Lancs.

O.M. Mercier, C., M.B., F.R.C.S., Flower House, Southend, Catford, S.E.

,, Mickle, W. J., M.D., F.R.C.P., Grove Hall Asylum, Bow, E.

,, Morris, Malcolm, F.R.C.S.Ed., 8, Harley Street, W.

1888 Mott, F. W., M.D , F.R.C.P., 84, Wimpole Street, Cavendish Sq., W. (Sec., 1894.)

1894 Murray, George, M.B., M.R.C.P., 2, Saville Place, Newcastle-on-Tyne.

1889 Murray, H. Montague, M.D., F.R.C.P., 27, Savile Row, W.

O.M. Nettleship, Edward, F.R.C.S., 5, Wimpole Street, W.

,, Nicolson, David, M.D., M.R.C.P.Ed., Broadmoor, Wokingham, Berks.

1888 Niermeyer, J. H. H., M.D., Amsterdam.

,, Norman, Conolly, M.K.Q.C.P., F.R.C.S.I., Richmond Asylum, Dublin.

1887 Ogilvie, George, M.B., M.R.C.P., 22, Welbeck Street, W.

1887 Ogilvie, Leslie, M.B., M.R.C.P., 46, Welbeck Street, W.

Oliver, J., M.D., M.R.C.P., 18, Gordon Square, W.C.

,, Oliver, Thomas, M.D., F.R.C.P., 7, Elison Place, Newcastle-on-Tyne.

O.M. Orange, Wm., C.B., M.D., F.R.C.P., 12, Lexham Gardens, Kensington, W.

,, Ord, W. M., M.D., F.R.C.P., 37, Upper Brook Street, W. (C. 1893.)

O.M. Ormerod, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W.

,, Page, Herbert William, M.A., F.R.C.S., 146, Harley Street, W. (C. 1891-2.)

,, Pasteur, Wm., M.D., F.R.C.P., 4, Chandos Street, W.

1888 Pitt, G. N., M.D., F.R.C.P., 24, St. Thomas's Street, S.E.

O.M. Poore, G. Vivian, M.D., F.R.C.P., 30, Wimpole Street, W.
1887 Pringle, J. J., M.B., F.R.C.P., 23, Lower Seymour Street, Portman Square, W.
O.M. Purves, W. Laidlaw, M.D., 20, Stratford Place, W.
„ Ramskill, J. S., M.D., M.R.C.P., 5, St. Helen's Place, E.C.
1894 Ransom, William B., M.D., M.R.C.P., The Pavement, Nottingham.
1890 Rayner, Henry, M.D., 2, Harley Street, W.
O.M. Reid, E. W., M.B., University College, Dundee.
1893 Rennie, George E., M.D., 46, College Street, Sydney, N. S. Wales.
1892 Richards, Joseph Peeke, M.R.C.S., 6, Freeland Road, Ealing, W.
1891 Rivers, W. H. R., M.D., M.R.C.S., St. John's College, Cambridge.
O.M. Romanes, G. J., F.R.S., 94, St. Aldate's, Oxford. (C. 1886-8.)
1892 Rowe, Edmund Lewis, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.
1892 Ruffer, Marc Armand, M.D., 5, York Terrace, Regent's Park, N.W.
1889 Russell, J. S. R., M.B., C.M., 4, Queen Anne Street, W.
O.M. Sanderson, J. Burdon, M.D., F.R.S., Banbury Road, Oxford.
„ Savage, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square. (C., 1886-90.)
1888 Savill, T. D., M.D., M.R.C.P., 12, Upper Berkeley Street, W.
O.M. Schäfer, E. A., F.R.S., University College, Gower Street. (V.P. 1892; P. 1893; C., 1886-90.)
„ Semon, Felix, M.D., F.R.C.P., 39, Wimpole Street, W.
„ Sharkey, S. J., M.D., F.R.C.P., 2, Portland Place, W. (C. 1889-92.)
1892 Shaw, James, M.D., M.Ch., Donald House, Kensington, Liverpool.
O.M. Sherrington, C. S., M.D., F.R.S., St. Thomas's Hospital, S.E. (C. 1892.)
1887 Smith, R. Percy, M.D., F.R.C.P., Bethlem Royal Hospital, Lambeth.
1892 Starling, Ernest Henry, M.D., M.R.C.P., B.S., 14, Grosvenor Road, S.W.
O.M. Stewart, T. Grainger, M.D., F.R.C.P.Ed., 19, Charlotte Square, Edinburgh.
„ Stirling, Wm., M.D., D.Sc., Owens College, Manchester.
1887 Suckling, C. W., M.D., M.R.C.P., 103, Newhall Street, Birmingham.
O.M. Sully, James, M.A., LL.D., 1, Portland Villas, Hampstead. (V.P. 1890-91.)
1889 Sutherland, Henry, M.D., M.R.C.P., 6, Richmond Terrace, Whitehall.
1888 Sykes, Henry, W., M.D., M.R.C.P., 3, Devonshire Street, Portland Place, W.
1891 Taylor, James, M.D., M.R.C.P., 34, Welbeck Street, W.
1889 Thorburn, Wm., F.R.C.S., Rusholme, Manchester.
1892 Titchener, E. B., B.A., Ph.D., 72, Heustis Street, Ithaca, N.Y.
O.M. Tooth, H. H., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891.)
1892 Trevelyan, E. F., M.D., B.Sc., 40, Park Square, Leeds.
1892 Tuckey, Charles Lloyd, M.D., 14, Green Street, Grosvenor Square, W.

O.M. Tuke, D. Hack, M D., F.R.C.P., 63, Welbeck Street, W. (Tr., 1889-93; V.P., 1894; C., 1887-8.)

1892 Tuke, J. Batty, M.D., F.R.C.P Ed., 20, Charlotte Square, Edinburgh

1891 Tuke, Thomas Seymour, M.B., Chiswick House, Chiswick.

1891 Turner, William Aldren, M.B., M.R.C.P., 13, Queen Anne Street, W.

O.M. Tweedy, John, F.R.C.S , 100, Harley Street, W.

1888 Voorthuis, J. A., M D., Medan, Deli, East Coast of Sumatra. (Communications to be addressed to M. Seyffardt, Bookseller, Amsterdam.)

O.M. Waller, Augustus, M.D , F.R S., 16, Grove End Road, N.W. (C., 1894)

„ Ward, J., D.Sc., Trinity College, Cambridge.

„ Warner, Francis, M D , F R C.P , 5, Prince of Wales Terrace, Kensington Palace, W.

1892 Washbourn, J W , M D., M.R.C.P , Guy's Hospital, S.E.

O.M. Watteville, A. de, M.A., M.D , B Sc., 30, Welbeck Street, W (C., 1890; Sec. 1886-9; Editor of *Brain.*)

„ White, W Hale, M D , F.R.C P., 65, Harley Street, W.

1889 Wiglesworth, Joseph, M.D., M.R C.P., County Asylum, Rainhill, Lancashire.

O.M. Wilks, Samuel, M D , F R.S., 72, Grosvenor Street, W. (Pres., 1887; V.P., 1886; C., 1888-91.)

1893 Wills, Ernest, M.D., M.R.C.P., Claybury Asylum, Woodford Bridge, Essex.

1893 Wood, Guy M., M.B., M.R.C.S., L.R.C.P , County Asylum, Rainhill, Lancashire.

1889 Wood, T. Outterson, M.D , M.R.C.P., 40, Margaret Street, Cavendish Square, W.

1892 Woodhead, G. Sims, M.D., F.R.C.P.Ed , Examination Hall, Victoria Embankment.

Elected in 1894.

Andriezen, W. Lloyd, M D., West Riding Asylum, Wakefield.

Cuming, James, M.D., F R.C.P I., 33, Wellington Place, Belfast.

Dean, H. Percy, M.S., F.R.C.S., 84, Wimpole Street, W.

Kidd, Percy, M.D., F.R C P., 60, Brook Street, W.

Martin, Sidney, M.D , F.R.C P., 10, Mansfield Street, W.

Shuttleworth, George E , M.D., Ancaster House, Richmond.

Walker, A. Stoddart, M.B., 13, Walker Street, Edinburgh.

Whiting, Arthur J., M.D., National Hospital, Queen Square, W C.

The Proceedings of the Neurological Society from its Foundation to the Year 1893.

The Neurological Society of London was founded in the year 1886, and the first General Meeting was held on January 14. As the Society publishes no transactions the Council have thought it advisable hereby to record its proceedings up to the present date, and in the future to publish a statement of the year's work with the Annual Report.

1886. March 24, Dr. J. Hughlings Jackson, F.R.S. Inaugural address, "On the Scope and Aims of Neurology."

May 15, Professors Schäfer, F.R.S., and Horsley, F.R.S., a demonstration on "Cerebral Localisation."

June 24, Dr. James Anderson, "A Case of Basal Cerebral Tumour affecting Left Temporo-sphenoidal Lobe." Mr. Jonathan Hutchinson, F.R.S., "A Case of Arrest of the Salivary Secretion (dry mouth)." Mr. Victor Horsley, (1) "Traumatic Epilepsy; Excision of Scar Involving Cortex;" (2) "Excision of Middle Division of Fifth Nerve for Neuralgia."

November 17, Dr. Savage, "The Relation of Epilepsy with Insanity" (Brain, vol. ix, p. 446).

December 16, Dr. Bastian, F.R.S., "The Muscular Sense, its Nature and Cortical Localisation" (Brain, vol x., 1), with discussion by Drs. Ferrier, Ross, Hughlings-Jackson, Mercier, de Watteville, Sir James Crichton-Browne; Profs. Sully, Horsley and Haycraft.

1887. January 27, Dr. Buzzard, "Two Cases of Thomsen's Disease" (*Lancet*, May 14, 1887). Dr. Ferrier, F.R.S., and Mr. Horsley, F.R.S., "A Case of Successful Removal of Cerebral Tumour." Dr. Hughes Bennett, "Chronic Epilepsy Successfully treated by Trephining" (*Brit. Med. Journ.*, 1887, i., p. 12). Dr. Beevor, "Cases of Infantile Paralysis, Paralysis Agitans and Tabes Dorsalis."

February 23, Dr. Hale White, "Case of Congenital Brain Disease." Dr. Ord, "Case of Chronic Cervical Pachymeningitis." Dr. Bristowe, F.R.S., (1) "Case of Rapid Action of the Heart;" (2) "Case of Unilateral Sweating." Dr. Donkin, "Case of Hammerman's Cramp." Dr. Hughes Bennett, "Compression of the Cord by Osseous Growth" Dr. Leslie Ogilvie, "Case of Supposed Disseminated Sclerosis in a child." Dr. Ormerod, "Cases of Muscular Atrophy" (*St. Barth's. Hosp. Reports*, vol. xxiii, p 104-106). Dr. Beevor, "Case of Tabes Dorsalis with Abductor Laryngeal Paralysis." Dr. Mitchel Bruce, "Tumour Compressing the Spinal cord" (Brain, vol. x., p. 210). Dr. Hughes Bennett, (1) "Sarcomatous Tumour of Medulla;" (2) "Tumour of Posterior Roots;" (3) "Limited Lesion of Internal Capsule, causing paralysis of one arm only during life."

March 26, Dr. SEMON and Mr. VICTOR HORSLEY, F.R.S., "A Demonstration on the Phonation Centre" (*Phil. Trans*, vol. clxxxi., 1890, B. p. 187). Dr. SANGER BROWN and Prof. SCHÄFER, F.R.S., "A Demonstration on the Sensory Centres."

May 12, Dr. GAY, "Ataxia of the Vocal Cords in Tabes Dorsalis." Dr. ORMEROD, "Case of Upper Arm Paralysis" (*St. Barth's. Hosp. Reports*, vol. xxiii., p. 90). Dr. BEEVOR, (1) "Cases of Atrophy of Muscles;" (2) "Cases of Disseminated Sclerosis" Dr. ORD, "Cases of Atrophy of Muscles." Dr. HADDEN, "Cerebral Tumour."

July 7, Dr. HUGHES BENNETT, Muscular Hypertonicity in Paralysis" (BRAIN, vol. x., 289). With Discussion by Drs. HUGHLINGS JACKSON, BUZZARD, FERRIER, DONKIN and HALE WHITE.

November 10, Dr. BRISTOWE, F.R.S., "A Case for Diagnosis." Dr. MOTT, "Sub-acute Myelitis of the Cervical Enlargement, resulting from a fall." Dr. BUZZARD, (1) "Paralysis of Fifth, Sixth, Seventh and Eighth Nerves;" (2) "Bulbar Symptoms in a Case of Insular Sclerosis." Dr. BEEVOR, (1) "Paralysis Agitans without Tremor;" (2) "Disseminated Sclerosis with Tabetic Symptoms." Dr. SAVILL, "Hysterical Paraplegia."

December 8, Dr. MERCIER, "Inhibition" (BRAIN, vol. xi., p. 361). With Discussion by Drs. HUGHLINGS JACKSON, WARNER, BROADBENT, SAVAGE and CATTELL.

1888. March 15, Dr. SHARKEY, (1) "Fatal Case of Tumour of Auditory Nerve" (BRAIN, vol. xi., p. 97); (2) "Case of Atrophy of Frontal and Parietal Lobes, consequent on prolonged Spinal Paralysis" (BRAIN, vol. xi., p. 94). Dr. BUZZARD, (1) "Sequel to a Case of Multiple Paralysis of Cranial Nerves" (BRAIN, vol. xi., p. 84); (2) "Case of Peripheral Neuritis with double wrist-drop, simulating Lead Palsy" (BRAIN, vol. xi., p. 90). Dr. HADDEN, "Ruptured Brachial Plexus." Drs HADDEN and SHERRINGTON, "Sections showing Ascending Degeneration in the Spinal Cord" (BRAIN, vol. xi., p. 325). Dr. HALE WHITE, "Brains of two rabbits in which Pyrexia had been produced by Lesions of the Corpus Striatum" (*Journ. of Physiol.*, vol. xi) Dr. A. DE WATTEVILLE, "Case of Aphemia."

May 5, Prof. SCHÄFER, F.R.S., "On the Visual Area of the Cortex" (BRAIN, vol. xi, p. 1). Dr. BEEVOR. Sections of Brain stained by Pal's modification of Weigert's method (lantern demonstration). Mr. E. P. FRANCE, A modified Weigert staining process. Degenerations following Lesions of the Marginal Gyrus.

June 28, Dr. W. H. GASKELL, F.R S., "The plan of Formation of the Spinal and Cranial Nerves" (*Med. Chi. Trans.*, vol. lxxi., p. 364). Prof. SCHÄFER, F.R.S., Photographs of the brains of monkeys shown to the Society on March 26, 1887, showing very Extensive Destruction of the Superior Temporal Gyrus and Temporal Lobe (BRAIN, vol. xi., p. 1 and p 145).

November 15, Dr. A. DE WATTEVILLE, (1) "Case of Analgesia in the Area of the Fifth Nerve;" (2) "Atrophic Paralysis of Arms and Neck;" (3) "Atrophic Paralysis with Increased Tendon Reaction." Dr. ORMEROD, "Peculiar Affection of Speech." Dr. J. HUGHLINGS JACKSON, F.R.S., "Case of Return of Knee-jerk in Tabes Dorsalis after Hemiplegia" (*Brit. Med.*

Journ., July 11, 1891). Dr. Ormerod, "Section, showing Combined Lateral and Posterior Sclerosis in which the Knee-jerk was present" (*St. Barth. Hosp. Reports*, vol. xxix.; figured in "Student's Guide to Nervous System," p. 216). Dr. James Anderson, "Case of Facial Hemiatrophy." Dr. Beevor, "Case of Traumatic Functional Contraction with Anæsthesia." Dr. Mitchell Bruce, "Case of Exophthalmic Goitre with General Muscular Atrophy." Dr. Hadden, "Case of Neuro-muscular Irritability" (Brain, vol. xii, p. 525).

December 20, Dr. Ferrier, "Cerebral Localisation in its Practical Relations" (Brain, vol. xii., p. 36).

1889. January, 24, Mr. J. Hutchinson, F R S., Inaugural address, "On the Nervous System in its Relation to Diseases of the Skin" (*Illus. Med. News*, 1889).

February 28, Mr. G. J. Romanes, F.R.S., "The Origin of Human Faculty" (Brain, vol. xii., p. 289).

May 18, Dr. Hale White, "The Experimental Production of Pyrexia in Rabbits by Lesions of the Corpus Striatum and its Neighbourhood" (*Journ. of Physiol.*, vol. xii.). Dr. H. B. Dean, "On an Experimental Investigation into the Variations in the Surface Temperature of the Two Sides of the Body in Compression of the Brain" (*Journ. of Path.*, 1894, p. 26). Mr. Victor Horsley, F.R.S, "On Clinical Observations during the past seven years on the Value of Differences Observed in the Temperature of the Two Sides of the Body, as Symptomatic of Cerebral Lesions."

June 25, Dr. Sherrington, "Degenerations following Lesions of the Motor Cortex" (a preliminary communication).

November 21, Dr. Hale White, "Case of Cured Cerebro-spinal Meningitis" (Brain, vol. xiii, p. 461). Dr. Taylor, "Case of Lead Paralysis." Dr. Hadden; (1) "Defect of Articulation in a boy;" (2) "Neuro-muscular Irritability" (a case shown November 15, 1888). Dr. Ormerod, (1) "Paralysis of Ocular Movements, with Spasmodic Affection of face, neck, speech, &c." (*Ophth. Soc. Trans.*, vol. iv., p. 310, and vol. xiii., p. 279); and "Sections of Spinal Cord from a case of Tabes Dorsalis, which terminated with symptoms of General Paralysis" (*St. Barth. Hosp. Reports*, vol. xxv., p. 87; figured in "Students' Guide to Nervous System," p. 208). Dr. Hughlings Jackson, "Case of Supposed Double Hemiplegia" (*Lancet*, vol. ii., 1892).

December 19, Dr Sully, "The Pscho-physical Process in Attention" (Brain, vol. xiii., p. 145).

1890. January 23, Dr. Buzzard, Inaugural address, "The Simulation of Hysteria by Organic Disease of the Nervous System" (Brain, vol. xiii, p. 1).

February 20, Drs. Russell and Taylor, "The Method of Treatment by Suspension, and its Therapeutic Value" (Brain, vol xiii., p. 206). Dr. Sherrington, "Results of Experiments in Suspension of the Cadaver" (Brain, vol. xiii., p. 449). Dr. Mitchell Clarke, Sections of the Spinal Cord in Tabes (Brain, vol. xiii., p. 356).

May 28, Dr F. W. Mott, "Results of Hemisection of the Spinal Cord in the Dorsal region."

June 26, Dr. Savill, (1) "Thickening of the Membranes of the Brain and Spinal Cord," (2) "Syringomyelia;" (3) "Hæmorrhage into Gyrus Fornicatus" (Brain, vol. xiv., p. 270); (4) "Neuritis in a phthisical patient." Dr. Maguire and Dr. Savill, Sections of Nerves from a case of Paralysis Agitans, showing Degeneration.

July 8, Dr. Savill, (1) "Cases of Muscular Atrophy with hereditary history" (*Nouvelle Iconographique de la Salpêtrière*, part 3, 1894); (2) "Varieties of Tremor." Dr. Donkin, "Muscular Atrophy in father and son" (Brain, vol. xiii., p. 456).

November 6, Dr. James Anderson, (1) "A Case of Morphœa, with Epilepsy;" (2) "A Case of Functional Spasm." Dr. Mitchell Clarke, Section of Spinal Cord from a case of complete Transverse Section at the level of the second Dorsal Root. Dr. Taylor, "Three Cases of Friedreich's Disease." Dr. Rivers, (1) "Locomotor Ataxy with Perforating Ulcer on each Foot, with Retention of Knee-jerks;" (2) "Case of Hemiplegia in Tabes with Return of the Knee-jerks on the paralysed side." Dr. May, "Paralysis of the Third, Fifth and Sixth Cranial Nerves."

December 18, Dr. Ross, "On Memory" (Brain, vol. xiv., p. 85).

1891. January 22, Dr. Bristowe, F.R.S., Inaugural address, "The Nature and Relation of Mind to Brain" (Brain, vol. xiv., p. 18).

March 5, Dr. Hadden, "Ataxic Movements in a child." Dr. Rivers, "A Case of 'Treadler's Cramp'" (Brain, vol. xiv., p. 110). Dr. J. Hughlings Jackson, F.R.S, "Return of both Knee-jerks in Tabes" (*Brit. Med. Journ.*, July 11, 1891). Dr. Hale White, "A Case illustrating the Diagnostic Value of Rise of Temperature in Hemiplegia" (*International Clinics*, vol. i., series iii., p. 162). Dr. Buzzard, (1) "A Case of Brachial Monoplegia;" (2) "Anomalous Tremors in a woman." Dr. Bristowe, (1) "Periodical Neuralgic Pain in the Arm attended with swelling, congestion and tenderness in the hand and forearm;" (2) A Case showing Attacks of Retraction of the Head and Neck, with Symmetrical Central Scotoma and Atrophy of the Optic Discs." Dr. Donkin, (1) "Functional Nervous Affection after Shock," (2) "A Case of Lead Palsy"

May 12, Dr. Beevor, "Some Points in the Actions of Muscles" (Brain, vol. xiv., p. 51). Dr. Waller, "On the Sense of Effort" (Brain, vol. xiv., p. 179).

June 12, Dr. Mott, "Hemisection of the Spinal Cord in monkeys" (*Phil. Trans.*, vol. clxxxiii., 1892, p. 1). Dr. Turner, "Hemisection of the Spinal Cord" (Brain, vol. xiv., p. 496).

November 19, Dr. Dupuy, "The Rolandic Area Cortex" (Brain, vol. xv., p. 190).

December 17, Dr. Savill, "Universal Hypertonicity of Muscles and Tendons in a male" (*Lancet*, 1889, vol. ii., p. 792). Dr. Turner, "Two Cases of Reflex Iridoplegia without Loss of Knee-jerks" (*Roy. Lond. Ophth. Hosp. Reports*, vol xiii., part 3). Dr. Ferrier, F.R.S., "A Case of Syringomyelia." Dr. Bristowe, F.R.S., (1) "Two Cases of Cerebellar Tumour;" (2) "Post-hemiplegic Hemichorea." Dr. Pitt, (1) "Spasm of the Ocular Muscles with Lingual Paralysis, and Tonic Spasm of the Extremities;" (2) "Symmetrical Paralysis in the Upper Extremities." Dr. Ormerod, "Progressive Muscular Atrophy Commencing in the Lower Extremities." Dr.

Galloway, "Sections of the Spinal Cord in Syringomyelia" (*Path. Trans.*, March 17, 1891).

1892. January 29, Dr. Bastian, F.R.S., Inaugural address, "The Neural Processes Underlying Volition and Attention" (Brain, vol. xv., p. 1).

March 17, Prof. Schäfer, F.R.S., "On Descending Degeneration following Lesions of the Cortex Cerebri" (partly published in "Quain's Anatomy," 1893). Dr. Mott, "On Ascending Degeneration following Lesions of the Spinal Cord" (Brain, vol. xv., p. 215). Dr. J. Risien Russell, "On the Arrangement of Nerve Fibres in Nerve Tracts and Roots in their Relation to Function" (*Phil. Trans.*, vol. clxxxiv., 1893, B. p. 39, and *Proc. Roy. Soc.*, vol. li., 1892, p. 22). Dr. W. H. Thompson, "Degeneration following Lesions of the Superior Temporal Gyrus." Dr. Tooth, "On the Ascending Antero-lateral Tract in the Medulla" (Brain, vol. xv., p. 397).

May 19, Dr. Savill, "Cerebral Tumour Accompanied by Anæsthesia of the Opposite Arm" (Brain, vol. xv., p. 448). Dr. Wigglesworth, "Pachymeningitis Interna Hæmorrhagica" (Brain, vol. xv., p. 431). Dr Ferrier, F.R.S., (1) "Tumour of Frontal Lobe;" (2) "Spinal Cord from a case of Syringomyelia." Dr. Taylor, (1) "Spinal Cord from a case of Syringomyelia" (*Lancet*, January 28, 1893); (2) "Two Cases of Tumour of the Spinal Cord with Ascending Degenerations."

June 16, Dr. Colman, "Paralysis of the Left Arm dating from infancy." Dr. Bastian, F.R.S, (1) "Unilateral Disseminated Sclerosis;" (2) "Athetoid Movements of the Left Arm" Dr. Buzzard, (1) "Multiple Neuritis following Influenza;" (2) Apoplectiform Bulbar Paralysis."

November 17, Dr. Head, "On the Distribution of Sensory Disturbances, with especial reference to the pain of Visceral Disease" (Brain, vol. xvi., p. 1).

December 15, Dr. Ferrier, F.R.S., "Syringomyelia." Dr. Pitt, "Muscular Atrophy with Hypertrophy of Neck Muscles." Dr. Mitchell Bruce, "Muscular Atrophy of Infantile Form." Dr. Bristowe, F.R S., (1) "Syphilitic Disease of the Nervous Centres, with Ataxia and other Symptoms." (2) "Hæmorrhage into, or Softening of, Pons Varolii." Dr. Eccles, "Crutch Palsy." Dr. Bastian, F.R.S., "Potts' Disease, with subsequent softening of the Spinal Cord, complete Motor and Sensory Paralysis, and almost complete Recovery" ("Hysterical or Functional Paralysis," 1893, p. 67). Dr. Wood, "Acute Spinal Disease, with Anomalous Symptoms."

1893. January 19, Professor Schäfer, F R S. Inaugural Address, "The Nerve Cell as the basis of Neurology" (Brain, vol. xvi., p. 134).

February 9, Dr. Sherrington, F.R.S., "The Skin Fields of some Sensory Spinal Nerve Roots" (*Phil. Trans.*, vol. clxxxiv., 1893. B, p 641).

May 11, Dr. Risien Russell, "On the Results of Cerebellar Lesions in Dogs" (*Brit. Med. Journ.*, 1893, and *Roy. Soc.*, not yet published). Professor Horsley, F.R S., and Dr. Kramer, "On an Experimental Investigation of the Effects of Gun Shot Wounds of the Brain" Dr. Mott, "The Effects of Removal of Cortical Motor Centres" (*Brit. Med. Journ.*, 1893, and *Journ. of Physiol.*, Jan., 1894). Dr. Hale White, "The Effects of Removal of 'the Arm Area' of the Cortex" (*Brit. Med. Journ.*, July 25, 1893). Dr. Sherrington, F.R.S., "Division of the Fifth Nerve" (*Journ of Physiol.*, vol. xiv, 1893, p. 255).

May 15, Mr. R. BOYCE, Photographs illustrating the degeneration in the Medulla and Cord following ablation of the Cerebral Hemispheres in Mammals. Dr. SHERRINGTON, F.R.S., Photographs illustrating the Skin Fields of the Upper Sensory Roots. Drs. BRUNTON, F.R.S., and CARRUTHERS, "Experiments in Elucidation of 'Treadler's Cramp.'" Dr. A. S GRÜNBAUM, "On the Local Fibres of the Spinal Segment." Professor SCHÄFER, F.R.S., and Dr. FLOOD, "Some Results of Extirpation of the Lower Part of the Ascending Frontal Gyrus."

June 15, Dr. J. HUGHLINGS JACKSON, F.R.S., "Ophthalmoplegia Externa with weakness of the Orbicularis Palbebrarum" (*Lancet*, July 15, 1893). Dr. FERRIER, F.R.S., "Ophthalmoplegia Externa, with weakness of the Orbicularis Palpebrarum." Dr. WOOD, "Tabes with Ophthalmoplegia, Paralysis of the Motor part of fifth nerve, and Laryngeal Paralysis." Dr. J. HUGHLINGS JACKSON, F.R.S., "Lesions of the Sixth, Seventh, and Twelfth Cranial Nuclei, with Paralysis of the Infra-hyoid Muscles." Dr. JUDSON BURY, "Persistent profound Hemianæsthesia, with marked Concentric Narrowing of the Visual Fields, and Trophic Lesions of the Hand on the affected side."

November 23, Dr. DONKIN, "A Case of Muscular Wasting of both Upper Extremities, and complete Paralysis of one." Dr. H LEWIS JONES, "Five Cases of Symmetrical Atrophy of the Intrinsic Muscles of the Hand in Young People" (*St. Barth's. Hosp. Reports*, vol. xxix.). Dr. ORMEROD, "A Case of Syringomyelia, showing loss of temperature sense, but not of pain." Dr. BEEVOR, "Paralysis Agitans, with Tremor of Lips, Tongue and Jaw." Dr. HECTOR MACKENZIE, "A Case of Friederich's Disease (*Amer. Journ. Med. Sci.*, 1894); A Case of Chorea in a man aged 81."